DOES NOT CIRCULATE

PUBLISHED WEEKLY

PRICE TWO SHILLINGS AND SIXPENCE

UNIVERSITY OF MICHIGAN
OCT 6 1954
MEDICAL LIBRARY

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—41ST YEAR SYDNEY, SATURDAY, SEPTEMBER 4, 1954 No. 10

COMMONWEALTH OF AUSTRALIA

DEPARTMENT OF HEALTH

STAPHYLOCOCCUS ANTI-TOXIN

(Globulins Refined and Concentrated)

Research has revealed that the staphylococcus may produce a variety of different toxins, each having destructive effect on some specific susceptible tissue. One toxin has a necrotic effect on the skin, another a destructive action on the red blood cells, another on leucocytes, and yet another a general lethal effect. Much of the destruction caused by staphylococci must be attributed to these toxins. These toxins are, immunologically, closely related, and are susceptible to the neutralizing action of Staphylococcus Anti-toxin. The administration of antitoxin is recommended in acute infections with toxæmia due to strains of Staphylococci (Staphylococcus Pneumonia, Osteomyelitis, and Septicæmia). Recent clinical experience has demonstrated the value of antitoxin in Staphylococcal Pneumonias of young children, in whom the toxæmia was not mitigated by antibiotics.

Commonwealth Serum Laboratories Staphylococcus Antitoxin available direct from the Serum Laboratories is standardized by comparison with the international standard of antitoxic activity.

Available in ampoules containing 5,000 *units and* 10,000 *units.*

COMMONWEALTH SERUM LABORATORIES
Parkville, N.2, Victoria, Australia

SL.58

Registered at the G.P.O., Sydney, for transmission by post as a newspaper.

NOW AVAILABLE!

CORTISONE ACETATE

***ORAL:** 5 mg. tablets: bottles of 50.
25 mg. tablets: bottles of 20 and 40.

***INJECTABLE:** Saline Suspension for intramuscular use.
25 mg. per c.c.: vials of 20 c.c.

TOPICAL: 1.5% Ointment: tubes of ½ oz.

OPHTHALMIC: 1.5% Cortisone Acetate combined with 0.5% Neomycin Sulfate in Andrew's "CORTOMYCIN" Ointment: tubes of ½ oz.

made in Australia

CORTISONE and HYDROCORTISONE preparations

HYDROCORTISONE ACETATE

ORAL: 20 mg. tablets: bottles of 25.

INJECTABLE: Saline Suspension for intra-articular use.
25 mg. per c.c.: vials of 5 c.c.

TOPICAL: 1% and 2.5% ointment: tubes of 5 grammes.

DENTAL: 2.5% ointment: tubes of 5 grammes.

OPHTHALMIC: 1% Hydrocortisone Acetate combined with 0.5% Neomycin Sulfate in Andrew's "HYDROCORTOMYCIN" Ointment: tubes of 5 grammes.

Further literature and information on request from the manufacturers

ANDREW'S LABORATORIES
15 HAMILTON STREET, SYDNEY

MANUFACTURERS OF DRUGS AND FINE CHEMICALS

**Prescribable according to Second Schedule (Restricted Drugs) of the P.B.A.*

AL.44.FP

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—41ST YEAR SYDNEY, SATURDAY, SEPTEMBER 4, 1954 No. 10

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— Page.

Obstetrics Today and Yesterday, by Lance Townsend 377
Volkmann's Ischæmic Contracture of the Forearm, by Colin Conacher, M.Ch.Orth., F.R.C.S. 383
Prevention of Hyaline Membrane After Cæsarean Section, by June Pash, M.B., B.S. 386
The Neuro-Pathology of the Landry-Guillain-Barré Syndrome, by R. D. K. Reye 386

REPORTS OF CASES—

Patent Ductus Arteriosus with Atypical Signs, by J. M. Gardiner 388

REVIEWS—

Practical Procedures in Clinical Medicine: Biochemical and Radiological Investigations 392
A Textbook for Midwives 393
A Study of Skeletons from a Medieval Danish Leper Hospital 393
The Psycho-Analysis of the Nursery 393
Diseases of Women 393
The Chest: A Handbook of Roentgen Diagnosis .. 394
Anoxia of the New-Born Infant 394
Bailey's Text-Book of Histology 394
Handbook of Tropical Dermatology and Medical Mycology 394
Surgery for Dental Students 395
Methods in Medical Research 395
Chemotherapy of Infections 395
Rorschach Responses in Old Age 395
The Mothercraft Manual 396
Entomology (Medical and Veterinary) 396

BOOKS RECEIVED 396

LEADING ARTICLES—

The Place of Biology in Education 397

CURRENT COMMENT—

Duration of Active Immunity Against Tetanus .. 398
Oxygen Therapy and Retrolental Fibroplasia .. 399
Donovanosis 400
The Chemotherapy of Orthopædic Tuberculosis .. 400
Polypi of the Rectum and Colon in Children 401
Neural Toxic Effects of Isoniazid 401

ABSTRACTS FROM MEDICAL LITERATURE— Page.

Bacteriology and Immunology 402
Hygiene 402

SPECIAL ARTICLES FOR THE CLINICIAN—

CVII. The Care of the Premature Infant 404

OUT OF THE PAST 410

CORRESPONDENCE—

The "British Medical Journal" and the British Medical Association in Australia 411
The Enigma of the Mona Lisa Smile 411
Volvulus of the Caecum 411
The Results of Surgical Treatment of Bronchiectasis 411
The College of General Practitioners Post-Graduate Week-End 412
Rubella (German Measles, Rotheln, Rubeola) .. 412

OBITUARY—

David Henry Edward Lines 412
Douglas John Thomas 413

POST-GRADUATE WORK—

The Post-Graduate Committee in Medicine in the University of Sydney 414

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 415

MEDICAL PRIZES—

The Norman McAlister Gregg Triennial Prize in Ophthalmology 415

NOTICE—

Physiotherapy Congress 416
Beattie-Smith Memorial Lectures on Insanity .. 416

MEDICAL APPOINTMENTS 416

NOMINATIONS AND ELECTIONS 416

DIARY FOR THE MONTH 416

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 416

EDITORIAL NOTICES 416

OBSTETRICS TODAY AND YESTERDAY.

By LANCE TOWNSEND,

Professor of Obstetrics and Gynæcology, University of Melbourne.

I AM aware of the honour that has been conferred on me in being asked to deliver this, the fourth R. H. Fetherston Memorial Lecture. I accepted the invitation with some diffidence, for each of the previous lecturers had been long established in practice at the time when he delivered his lecture, and each had had the inestimable advantage of knowing Dr. Fetherston personally.

Dr. Fetherston was interested in the problems of maternal mortality and morbidity, and it was his wish that these lectures should be given from time to time so that obstetric practice in Victoria could be brought under critical review and the medical and nursing professions would be informed of present-day trends. It is well known that the practice of obstetrics is not static. How many of us realize how rapidly changes are occurring?

If Dr. Fetherston were able to revisit the labour ward at the Women's Hospital today, he would be amazed at the changes that have taken place. He would first notice that the labour ward was a much quieter place. Secondly, if he stayed for a while, he would not see the drama that used to exist from time to time when he worked in the hospital. In those days an obstetrician was judged chiefly on his ability and skill as a manipulator; the emphasis today is on the prevention of obstetric situations which require difficult manipulations as the method of treatment.

In my lecture tonight I propose to compare obstetric practice in 1943 with that in 1953. I will take the methods used at the Women's Hospital, Melbourne, as an example of obstetric practice in these years and compare the results of these methods by a statistical analysis.

Dr. Fetherston would have approved of my choice of hospital as an example of obstetric practice, as he was born at the hospital, worked in it, and to his death maintained an active interest in it.

I have called my lecture "Obstetrics Today and Yesterday", for you and I live in an atomic age when ten years ago is like yesterday. In comparing obstetric practice in 1943 with that in 1953, I intend first of all to consider maternal deaths and then to discuss in detail the major causes of death: toxæmia of pregnancy, rupture of the uterus and Cæsarean section, post-partum hæmorrhage and puerperal infection. In each of these conditions changes in treatment will be discussed and results assessed. The fœtal statistics will then be considered. The annual clinical report of the Women's Hospital is compiled for twelve months ending on June 30 of each year. In this lecture, when figures for 1943 are discussed they are for the period from July, 1942, to June, 1943, and similarly for 1953. In these statistics two types of cases are described. A "booked" patient is a patient who has attended the ante-natal clinic at least twice before her admission to hospital. All other cases are classified as emergencies.

[1] Delivered on April 7, 1954.

In the two years under review the number of patients delivered at the hospital was as shown in Table I.

It is seen that in the last ten years the number of booked patients has more than doubled, while the number of emergency patients has remained about the same.

TABLE I.
Patients Delivered.

Type of Patient.	1942–1943.	1952–1953.
Booked	2702	6301
Emergency	639	501
Total ..	3341	6802

Maternal Deaths.

In 1943 there were 20 maternal deaths, in 1953 there were five. This shows a substantial reduction and indicates that there is a general trend towards a lowering of the maternal mortality rate. In the booked cases the reduction is from 1·85 per 1000 to 0·32 per 1000. In the emergency cases the reduction is from 23·5 per 1000 to 6·0 per 1000. It is of significance that the improvement is just as much in the emergency cases as in the booked cases.

TABLE II.
Maternal Deaths.

Deaths.	1942–1943. (3341 Deliveries.)	1952–1953. (6802 Deliveries.)
In booked cases	5 (1·85 per 1000)	2 (0·32 per 1000)
In emergency cases	15 (23·5 per 1000)	3 (6·0 per 1000)
Total	20 (5·99 per 1000)	5 (0·73 per 1000)

By a consideration of the causes of death in each year it is possible to show in which condition there has been a reduction in the number of deaths.

TABLE III.
Causes of Death.

Condition.	1942–1943.	1952–1953.
Eclampsia	5	1
Preeclampsia	1	1
Nephrosis	3	—
Ruptured uterus	3	—
Post-partum hæmorrhage ..	2	—
Septicæmia	3	—
Pneumonia	1	—
Pulmonary tuberculosis ..	1	—
Sarcoma of duodenum ..	1	—
Cerebral tumour	—	1
Diabetic coma	—	1
Virus infection (generalized)	—	1
Total	20	5

The number of deaths from the toxæmias of pregnancy has been reduced from nine to two, that from rupture of the uterus from three to nil, that from post-partum hæmorrhage from two to nil, and that from puerperal infection from three to nil. Each of the major causes will now be considered in greater detail.

Toxæmias of Pregnancy.

The reduction in the number of deaths from toxæmia of pregnancy has been the greatest from both an absolute and a relative point of view. I consider that this is due to the changes in the treatment of this condition.

When Arthur Wilson delivered the first R. H. Fetherston Memorial Lecture in 1945, he stated that the obstetricians at the Women's Hospital had had promising results by using magnesium sulphate in the treatment of eclampsia. He urged all practitioners to use this drug in the treatment of the disease, and it is significant that today it still has a place in its management.

Ivon Hayes, in the second Fetherston lecture in 1948, said that toxæmia of pregnancy could be avoided or deferred by proper dietetic control. Although obstetricians had used this prophylactic method in their private practice, it was not until three years ago that these methods were applied universally to all patients attending the ante-natal clinics at the Women's Hospital. There are three essential principles which must be carried out if this method is to be a success. Firstly, a strict supervision of all ante-natal records is maintained so that patients who do not attend as instructed are sent for. Secondly, patients are carefully weighed at each visit. Thirdly, detailed dietetic instructions are given. Any patient gaining more than three pounds in four weeks is put on a diet of high protein and low Calorie content with added vitamins. The diet is varied according to weight changes so that there is a total gain during pregnancy of about 21 pounds. Failure to attend after a period of excessive gain in weight must be noted and the patient sent for, as it is in these cases of default that toxæmia of pregnancy is apt to occur. Although it is not possible to give comparable figures for the incidence of severe preeclampsia for 1943 and 1953, owing to a change in classification of the disease, a clear-cut indication of the improvement is seen from a study of the maternal and fœtal deaths. In 1943 the diagnosis of preeclampsia was not employed until albuminuria appeared; in 1953 the presence of generalized œdema and a relative rise in blood pressure beyond 20 millimetres of mercury, systolic, or 10 millimetres, diastolic, are sufficient for this diagnosis, even in the absence of any albumin in the urine. In 1943 four mothers died from preeclampsia, and in 1953 one died. In 1943 the fœtal wastage—that is, the number of fœtal deaths—was 39; in 1953 it was 52, a reduction from 11·5 to 7·4 per 1000.

TABLE IV.
Preeclampsia.

Deaths.	1942–1943. (3341 Deliveries.)	1952–1953. (6802 Deliveries.)
Maternal	4	1
Fœtal	39 (11·5 per 1000)	52 (7·4 per 1000)

TABLE V.
Eclampsia.

Observation.	1942–1943.	1952–1953.
Number of booked patients	9	9
Number of emergency patients	40 } 49	19 } 28
Materna deaths	5 (10·0%)	1 (3·5%)
Fœtal results:		
Stillbirths	16	1
Neonatal deaths	5	1
Fœtal wastage	21 (43·0%)	2 (7·2%)

The statistics for eclampsia are more complete and show in a more satisfactory way the changes that have occurred.

There were no maternal deaths in either year from eclampsia amongst booked patients. In emergency cases there were five maternal deaths in 1943 and one in 1953. *The maternal death rate has dropped from 10% to 3·5%.* The usually accepted death rate for eclampsia is 10% to 15%; the figure for 1953 is well below this.

The incidence of the disease in both booked and emergency cases has also been reduced.

In booked cases during 1943 the incidence was nine of 2702 cases; in 1953 it was nine of 6301 cases. This is a *reduction of 50%.* Likewise there was a reduction in the incidence of the disease in emergency patients from 40 of 639 admissions in 1943, to 19 of 501 admissions in 1953. *This decrease in the number of admissions implies that the* incidence of the disease in the metropolitan area has become less and is probably due to the use of improved prophylactic methods. *In 1943 there were 21 fœtal deaths;* in 1953 there was one. This is a reduction from 43% to 7%. *This remarkable fall is due to the decreased severity of the* disease. In this decade in patients suffering from eclampsia there has been a threefold change. There has been a *reduction in the incidence, in maternal death rate and in* fœtal death rate.

These results are very encouraging and justify the time spent in making sure that patients attend regularly, that they are weighed, that the dietetic service is utilized and *that the dietetic régime is followed. There is, of course,* a great deal more to the treatment of toxæmia of pregnancy than control of the weight.

I consider that the widespread acceptance by obstetricians in Victoria of the principle that by correct nutrition the incidence and *severity of toxæmia of pregnancy can be* reduced has been one of the major advances in the decade.

Rupture of the Uterus and Cæsarean Section.

In 1943 rupture of the uterus occurred in six cases and resulted in three maternal deaths. In 1953 it occurred in four cases without any maternal deaths. The causes of *rupture in 1943 were as follows: neglected transverse* lie of the fœtus in three cases, internal version for brow presentation in two cases, and in one case multiparity (in a patient who had previously had eight children). In 1953 the causes were as follows: manual removal of adherent placenta in two cases, neglected transverse lie of the fœtus in one case, and internal version in one case.

TABLE VI.
Rupture of the Uterus.

Observation.	1942–1943. (3341 Deliveries, 6 Cases.)	1952–1953. (6802 Deliveries, 4 Cases.)
Maternal deaths	3	—
Stillbirths	6	4
Preceding factors:		
Neglected transverse lie of fœtus	3	1
Internal version for brow presentation	2	1
Multiparity	1	—
Manual removal of placenta	—	2

Factors that produce rupture of the uterus repeat themselves from year to year, so that a significant reduction in the incidence of any one cause indicates an improvement in the method of treatment of that cause. The reduction of the maternal death rate from three to nil has been made possible by improvement in the surgical ancillary services —namely, availability of blood, modern anæsthesia and chemotherapy. Although modern treatment will generally save the mother's life, the condition is almost uniformly fatal for the baby.

Cæsarean Section.

Let us next consider Cæsarean section. In 1953 there were no deaths among patients delivered by Cæsarean section. In 1943 one patient died on the operating table *whilst a Cæsarean hysterectomy was being performed for a* ruptured uterus, and another patient died from general peritonitis ten days after a classical Cæsarean section and suture of the uterus had been performed for a ruptured uterus.

The indications for Cæsarean section as a method of treatment in obstetric practice are always under discussion, and I propose to outline some of the changes which may have occurred in the last decade. The incidence of Cæsarean

TABLE VII.
Cæsarean Section.

Observation.	1942–1943. (3341 Deliveries.)	1952–1953. (6802 Deliveries.)
New cases	51 } 64 (1·91%)	127 } 183 (2·69%)
"Repeat" cases	13 }	56 }
Maternal Deaths	2	—

section in the treatment of some obstetrical complications *is increasing.* It is of interest that Arthur Wilson in 1945 stated that Cæsarean section should be notifiable because of the high maternal death rate associated with it. Although *the maternal death rate is much lower now, it is important* that students are told that, although Cæsarean section may appear to be the easy way out of an obstetrical problem, it is not always the safest for mother or baby.

TABLE VIII.
Types of Cæsarean Section.

Type of Operation.	1942–1943.	1952–1953.
Classical Cæsarean section	13	8
Lower segment Cæsarean section	47	174
Cæsarean hysterectomy	4	1

The incidence of the operation in *1943 was 1·91%; in* 1953 it was 2·69%. This is an increase of 40%. The *present rate of 2·69%* is still a low figure on world standards, and is one of which we are proud, because it is associated with an equally low fœtal death rate. The two *maternal deaths that occurred in 1943 have already been* discussed under the heading of rupture of the uterus, and in both cases the patient died from other causes.

TABLE IX.
Reasons for Performance of Cæsarean Section.

Reasons for Cæsarean Section.	1942–1943. (51 Cases.)	1952–1953. (127 Cases.)
Cephalo-pelvic disproportion	30	30
Abnormal uterine action	—	7
Placenta prævia	12	31
Preeclampsia	1	9
Failed artificial rupture of membranes for preeclampsia	—	4
Chronic nephritis	2	1
Prolapsed cord	—	6
Previous Manchester repair	1	5
Breech presentation	—	5
Rupture of uterus	3	1
Miscellaneous	2	28

Over the years, there has been a gradual change in the type of operation performed. In 1943 20% of all Cæsarean sections were of the classical type; in 1953 4% were of this type. The use of the classical operation has gradually diminished, and now the indications for this operation are few. From a consideration of the indications for the operation in both years any changes that may have occurred can be ascertained.

The first change is that the number of indications has increased, and for each indication there is an increase in the number of cases. The major exception to this latter increase is cephalo-pelvic disproportion. This was the most common indication in 1943. In 1953 it was not so; the change is due partly to the fact that some cases previously classified as disproportion are now placed under the heading of abnormal uterine action, and partly to the wider use of X-ray pelvimetry in the diagnosis of contracted pelvis.

The next change to be noted is the increase from 12 to 32 in the number of cases of *placenta prævia* treated by Cæsarean section. As well, the type of Cæsarean section used has changed. In 1943 the classical operation was the method of choice; in 1953 it was the lower segment operation. This is an important change in treatment, for subsequent deliveries may take place *via* the vaginal route if the scar in the lower segment of the uterus is presumed to be satisfactory. The fœtal death rate has been reduced

TABLE X.
Placenta Prævia.

Observation.	1942–1943.	1952–1953.
Number of cases	41	59
Number of patients treated by Cæsarean section	12	32
Type of operation:		
Lower uterine segment	2	31
Classical	10	1
Fœtal results:		
Stillbirths	16	9
Neonatal deaths	2	6
Total wastage	18	15

from 18 out of 41 to 15 out of 59. This has not been achieved solely by increased use of Cæsarean section; two other factors have contributed. Firstly, blood is now readily available, and it has saved the lives not only of mothers, but of babies as well, for in this condition the baby often dies *in utero* because of anoxia. Secondly, patients are now treated conservatively for as long as possible in an effort to obtain a more mature baby. This is achieved by keeping the patient in hospital and by having a blood transfusion service available. These factors have reduced the fœtal death rate, but the figure still remains high. Part of this high death rate is due to circumstances which are difficult to control. Firstly, the fœtus may die during a severe hæmorrhage before treatment can be instituted, and, secondly, the baby may subsequently die of anæmia and prematurity when a severe hæmorrhage occurs in the early part of the third trimester and a Cæsarean section is necessary to control the bleeding and so save the mother's life.

The third change in the indications for Cæsarean section is an increase in its use for the treatment of preeclampsia. I am very hesitant to perform a Cæsarean section in cases of preeclampsia. Conservative treatment along the usual lines with reduction of the blood pressure by hexamethonium compounds will usually stop the patient from developing eclampsia and make a vaginal delivery safe.

Indications in 1953 which do not appear in 1943 are the following: prolapse of the cord, failure to induce labour after artificial rupture of the membranes, complicated breech presentation and previous Manchester repair.

These statistics show that Cæsarean section today can be a safe operation. This is due to many conditions and factors. These are as follows: the usual incision is in the lower segment of the uterus, skilled anæsthesia is available, chemotherapy is used when required, and blood can be obtained. When these conditions are not all fulfilled the operation should be performed only as an emergency.

In 1943 the total fœtal death rate among babies delivered by Cæsarean section was 21·9%; in 1953 it was 12·6%. The 1953 figure compares favourably with the best figures from other parts of the world. When the fœtal statistics in new cases are considered, the fœtal deaths are usually due to the obstetric condition that required treatment by Cæsarean section rather than to the operation itself. In "repeat" cases in which Cæsarean section is performed as an elective operation, conditions should be optimal as far as the baby is concerned; and yet in 1953 there were four neonatal deaths in 56 cases—a neonatal death rate of 7·2%. The cause of death in these cases was atelectasis in two and hyaline membrane in two. It has been suggested that if the membranes are ruptured a few hours before the elective Cæsarean section is performed, the incidence of hyaline membrane in the baby is reduced. This method is being tested at the hospital at present.

TABLE XI.
Cæsarean Section: Fœtal Statistics.

Type of Case.	1942–1943.	1952–53.
New cases:		
Number of cases	51	127
Stillbirths	10	8
Neonatal deaths	3	11
Fœtal wastage	13	19
"Repeat" cases:		
Number of cases	13	56
Stillbirths	—	—
Neonatal deaths	1	4
Fœtal wastage	1	4

I am often asked which is the safer method by which to have a baby. For the mother, provided there are no complications present, the vaginal route is definitely the safer. This is also true for the baby. It used to be stated: "Once a Cæsarean, always a Cæsarean." In 1943, of the 19 pregnant patients who had previously been delivered by Cæsarean section, 13 were treated by an elective Cæsarean section and six had a trial of labour which resulted in a vaginal delivery. In 1953, of the 95 similar patients, 56 had an elective Cæsarean section and 39 had a vaginal delivery. The change of policy in this type of case is due to the commoner use of the lower segment operation. In 1943 31% of the patients delivered themselves *via* the vaginal route, and in 1953 41% delivered themselves this way. In both groups there were no cases of rupture of the uterus. In 1943 two babies were lost, and in 1953 there were also two fœtal deaths—one due to erythroblastosis and the other due to anoxia occurring before the onset of labour.

Despite these results, care must be exercised in the selection of patients for trial of labour as a method of treatment of patients who are pregnant and who have been delivered previously by Cæsarean section. With the increase over the last ten years in the use of Cæsarean section as a method of treatment in selected cases, the maternal mortality has remained stationary and the fœtal mortality has been reduced.

TABLE XII.
Post-Partum Hæmorrhage.
(Loss of One Pint or More.)

Observation.	1942–1943. (8341 Deliveries.)	1952–1953. (6802 Deliveries.)
Number of cases	411	155
Rate per 100 cases	11	2·2

Post-Partum Hæmorrhage.

Post-partum hæmorrhage accounted for two maternal deaths in 411 cases in 1943, and for no deaths in 155 cases in 1953. The reason for the decrease in the number of deaths and for the change in incidence of the condition from 11% to 2·2% is threefold. Firstly, if a post-partum hæmorrhage occurs, the amount of blood lost is correctly measured and is not reduced to a minimum by the midwife.

Secondly, blood is available for these patients. Thirdly, hæmorrhage is reduced by the use of 0·5 milligramme of ergometrine given intravenously after the second stage of labour is completed. The use of an ergot derivative before the placenta is delivered is certainly a great break with tradition and one which is not universally agreed on by all obstetricians.

Since ergometrine has been used in this way at this hospital, great changes have been noticed in the labour ward. In 1943 there were commonly patients in the labour ward receiving a blood transfusion as part of the treatment of a post-partum hæmorrhage, or suffering from shock due to a post-partum hæmorrhage. In 1953 very few of these patients were seen, as the incidence of post-partum hæmorrhage has been reduced. The rapid clearing of patients from the labour ward is of great help, as the ward was built to deliver 3000 patients and not 7000 *per annum*. The other great advantage is that less blood is being used.

With the routine intravenous use of ergometrine the incidence of retained placenta has increased. In 1953 there were 157 cases of retained placenta. These patients were treated by manual removal of the placenta. In all cases after the placenta is delivered 0·5 milligramme of ergometrine is given intramuscularly.

Care must be exercised in the use of ergometrine in the treatment of patients with twins. If the injection is given after the first twin is born, then the second twin may be expelled rapidly from the uterus. If, however, the lie of this baby is not longitudinal, then it may be retained inside a tonically contracted uterus. This results in a stillbirth.

Despite the complications mentioned above, the use of ergometrine after the birth of the baby has been one of the major advances in this decade. Post-partum hæmorrhage, which used to be the nightmare of all obstetricians, should now be an infrequent occurrence in any obstetric practice.

Puerperal Infection.

Puerperal infection is the last of the major causes of death, and as in the former causes there has been a reduction in the death rate. In 1943 five patients died from puerperal infection. In 1953 no patients died. There is no need for me to state why this improvement has taken place. We are all aware of the place of chemotherapy and antibiotics in the treatment of this disease.

In 1943 two patients died from septicæmia, one died from acute bacterial endocarditis, and two died from general peritonitis complicating a rupture of the uterus. In 1953 one death was due to a severe virus infection which involved the heart, lungs, liver and kidneys. This death reminded us that, although we may have much of the bacterial world under control, the filtrable virus has still to be conquered. This patient was found dead in her bed twelve hours after a normal delivery. A similar infection has occurred this year, the patient having died undelivered. However, the death rate alone does not tell us the whole story of infection.

It is necessary to consider the morbidity rate. In 1943 the morbidity rate was 8·9%. In 1953 it was 1·5%. This reduction is significant and indicates that the present methods of treatment, which I shall mention later, are in the main adequate. This lowered incidence of morbidity means that more patients are now discharged from hospital in a good state of general health, with adequate lactation, and facing their added responsibilities with confidence.

There has been no change in the proportion of infections occurring in genital and extragenital situations. In the extragenital types the incidence and severity of acute mastitis in the puerperium have fallen considerably. This is due to the early prophylaxis and treatment of the condition by antibiotics. The reduction in the incidence of this complication has increased the percentage of mothers who breast-feed their babies.

In the 73 cases of infection that occurred in 1953, the type of organism follows a pattern that is peculiar to Melbourne. Here the major bacterial cause of puerperal infection is the anærobic streptococcus. All the 73 patients, with the exception of the one who had an infection with *Clostridium welchii*, were treated with success by penicillin and mixed sulphonamides. As far as puerperal infections are concerned, the newer antibiotics are very rarely needed to cure the condition.

At present infection as a cause of death in childbirth and as a cause of morbidity is not a major factor. This

TABLE XIII.
Puerperal Infection.

Observation.	1942–1943. (8341 Deliveries.)	1952–1953. (6802 Deliveries.)
Maternal deaths	5	—
Number of cases of infection	309	104
Morbidity rate	8·9%	1·5%
Site of infection:		
Genital	210	73
Extragenital	99	31

does not mean that we can be complacent; on the contrary, we must be more careful than we were, for the students and midwives now being trained have never seen the ravages of puerperal infection and wonder why so much care is taken to prevent it. They say that surely this care is unnecessary. The aseptic ritual in the labour wards must be rigidly observed. In addition, many strains of

TABLE XIV.
Puerperal Infection.

Type of Organism.	Number of Cases.
Anaerobic streptococci	38
Hæmolytic streptococci group A	13
Bacterium coli	8
Staphylococcus pyogenes	6
Anaerobic non-hæmolytic streptococci	1
Group D streptococci	2
Clostridium welchii	1
No organism found	4

bacteria are becoming resistant to known antibiotics, and we may again in the future find ourselves without the effective compounds that we have today to combat the disease.

Septic Abortion.

Let us now consider infections occurring before viability of the fœtus. The main type is septic abortion.

TABLE XV.
Septic Abortion.

Observation.	1942–1943.	1952–1953.
Total number of cases	478	250
Number of deaths	26 (5·4%)	3 (1·2%)

In 1943 there were 478 cases, in 1953 there were 250 cases. During the same period the number of "clean" abortions rose from 1102 to 1400 cases, and it is reasonable to assume that "septic" cases would rise in a similar proportion. This has not occurred, and in fact the number of cases has been halved. Added to this, the mortality from the condition has dropped from 5·4% in 1943 to 1·2% in 1953. These changes in the incidence and in the death rate are very welcome. I have always regarded these deaths as unnecessary, and although one would like to think that the reduction was due to better education

regarding the risks of criminal abortion, it is clear that chemotherapy and the antibiotics have played the major part in this significant change.

Conclusion.

I have quoted statistics from the Women's Hospital to illustrate the results of obstetric practice at that hospital, and have indicated the major changes in treatment which I consider have contributed to the improvement. I am now going to show that there has also been an improvement throughout the State by quoting from the vital statistics for Victoria. (Note that the State figures are for a calendar year.)

TABLE XVI.
Victorian Vital Statistics.

Observation.	1943.	1953.
Number of viable infants delivered	39,100	53,500
Maternal deaths	136	33
Maternal death rate	3·48 per 1000	0·62 per 1000

It is significant that the maternal death rate for Victoria has dropped from 3·48 per 1000 in 1943 to 0·62 per 1000 in 1953. As with the hospital figures, a consideration of the actual causes of death will show in which conditions the improvement has taken place.

TABLE XVII.
State of Victoria: Maternal Deaths.

Cause of Death.	Number of Cases.	
	1943.	1953.
Post-abortional sepsis ..	44	1
Puerperal sepsis	23	2
Toxæmia	30	8
Accidents of child-birth ..	10	5
Ectopic gestation	9	—
Abortion, not septic ..	6	1
Ante-partum hæmorrhage ..	3	—
Post-partum hæmorrhage ..	9	2
Cardiac diseases	—	7
Intercurrent disease	—	3
Pulmonary embolus	—	2
Other diseases	2	2
Total	136	33

Here again there has been a reduction of deaths from the major causes—namely: toxæmia of pregnancy from 30 to eight cases; rupture of the uterus and Cæsarean section from 10 to five cases; post-partum hæmorrhage from nine to two cases; puerperal infection from 23 to two cases; and post-abortional sepsis from 44 cases to one case.

These figures indicate that throughout the State there has been an improvement in the care and treatment of pregnant women.

Let us now consider the fœtus—a life just as important as that of the mother.

The number of fœtal deaths in 1943 was 87 per 1000 live or stillborn babies; in 1953 it was 36 per 1000. This is a 100% improvement. The stillbirths dropped from 52 to 20 per 1000 and the neonatal deaths from 35 to 16 per 1000.

The reduction in the stillbirth rate and the neonatal death rate is due to a combination of factors. Better obstetric care during pregnancy has been one factor, close liaison between obstetrician and pædiatrician another, and improvement in the methods of treating the sick newly born by pædiatricians yet another.

The fœtal death rate of 28 per 1000 in "booked" cases compares favourably with any published reports from other centres.

The Women's Hospital honorary staff are still not satisfied with these figures, and it is pleasing to record that for the nine months of the present year ending June, 1954, the fœtal death rate for "booked" cases is under 20 per 1000. In absolute numbers, the reduction in the fœtal death rate over the ten years represents a large saving of babies' lives.

From the statistics there has been a reduction in both maternal and fœtal death rates. If these rates had remained stationary in 1953, 35 more mothers and 330 more babies would have died. The saving of these lives indicates that the changes and developments in treatment have been correct.

Dr. Arthur Wilson concluded his R. H. Fetherston Memorial Lecture with a seven-point challenge, and Dr. Ivon Hayes reemphasized the first two of these points. Tonight on behalf of my colleagues of the Women's Hospital I take up the challenge. I believe that in the last ten years all Dr. Wilson's points have been answered in part or in full. What were these points?

TABLE XVIII.
Women's Hospital: Fœtal Statistics.

Observation.	1942–1943.	1952–1953.
Number of babies born ..	3391	7026
Number of stillbirths ..	176	141
Rate per 1000	52	20
Neonatal deaths	117	115
Rate per 1000	35	16
Total wastage	293	256
Rate per 1000	87	36

Dr. Wilson wanted a Consultative Council on Maternal Mortality set up by the State Government. This Council would consider and advise on all maternal deaths occurring in the State. This Council has been formed and has been carrying out the above-mentioned functions for over one year.

Dr. Wilson's next point was that the Council should issue reports from time to time to the medical profession; these reports were to include a summary of the deaths that had occurred and any new advance in diagnosis and treatment in obstetrics. These reports will be issued, and already one has been circulated on the subject of early diagnosis and treatment of toxæmia of pregnancy.

Dr. Wilson's third point was that an attempt should be made to reduce the number of criminal abortions and subsequent maternal deaths. It is doubtful whether there has been any reduction in the incidence of criminal abortions; but there has been a dramatic decrease in the number of deaths from their septic complications.

Dr. Wilson's next point was that he wished all Cæsarean sections and cases of eclampsia to be notifiable. His reason for this was that in 1945 both conditions carried a high maternal mortality. Over the last ten years the death rates from Cæsarean section and eclampsia have both fallen considerably, and if medical practitioners will follow the principles of treatment as laid down, the maternal mortality from these two factors will be kept to a minimum.

Dr. Wilson's fifth point was that all stillbirths should be registered; legislation recently passed has made this compulsory.

Finally, Dr. Wilson wanted post-graduate courses instituted for nurses and doctors and increased facilities for teaching of obstetrics to medical students. Post-graduate courses for medical practitioners are held twice a year at the Women's Hospital, and lectures are given by the Fellows and Members of the Royal College of Obstetricians and Gynæcologists from time to time. The Royal Victorian College of Nursing and the College of Nursing, Australia, provide post-graduate courses for midwives, and this year the latter body has instituted a course for sister-tutors in midwifery. The University of Melbourne has set up a professorial unit at the Women's Hospital, and I think that

the teaching of obstetrics to medical students by the honorary medical staff and the professorial unit at the Women's Hospital is satisfactory.

From a survey of the statistics I am sure you will agree that the practice of obstetrics has improved over the last ten years. This progress has been due, not to any one obstetrician, but to many, and I should be wrong to name any person in particular, for many have made a contribution in a quiet way. Obstetricians and pædiatricians now work as a team, building on the sound foundations laid down by our predecessors.

We are here tonight to commemorate one of the leading men of the past who contributed to the progress of obstetrics. Were Dr. Richard Herbert Fetherston to revisit the Women's Hospital today, I think he would be proud of the institution in which he was born. He would say to us, who are working in the hospital at the moment: "Your results are good, but there is still room for improvement. Be ever careful, diligent and kind to your patients; you all have a contribution to make." That Dr. Fetherston thought along these lines is evident from the fact that this lecture tonight is one of a series endowed by him in perpetuity. His aim, like ours, was that every mother should be safely delivered of a normal baby, which she could rear in health and happiness. "The race is not to the swift nor the battle to the strong. But time and chance happeneth to them all."

VOLKMANN'S ISCHÆMIC CONTRACTURE OF THE FOREARM.

By COLIN CONACHER, M.Ch.Orth., F.R.C.S.,
Sydney.

VOLKMANN'S CONTRACTURE is a paralysis of the fingers leading to deformity, following trauma to the region of the elbow and forearm. It is a condition poorly described in the literature, although considerable progress has been made in recent years both in understanding and in treating it. Severe cases are rare, but mild cases are not uncommon, and are sometimes not recognized, being confused with post-traumatic finger stiffness. I have been able to find 22 old and new cases in eight hospitals in two years.

The characteristic deformity consists of flexion of the interphalangeal joints of the fingers, which increases with extension of the wrist; in severe cases there is also flexion of the elbow, pronation of the forearm and flexion of the wrist.

The commonest preceding injury is a supracondylar fracture of the humerus, fractures of both bones of the forearm being almost as frequent. Practically any injury of the elbow or forearm may lead to Volkmann's contracture, and it has been known to follow fracture of the clavicle, embolism of the brachial artery, and compression injury of the forearm without bone damage. It is most often seen in children, probably because fractures of the elbow joint and forearm are commonest in childhood; but it may occur in adults.

Pathology and Causes.

The usual muscles to be affected are the *flexor digitorum profundus* and the *flexor pollicis longus*. Other flexors in the forearm, the pronators and the brachialis, are also involved in severe cases. Rarely it may affect the forearm extensors only. In the hand the muscles may show paralysis and contracture, but this is due to denervation and not to ischæmic myositis. This condition of the hand associated with ischæmic paralysis of the forearm muscles is quite distinct from a rare type of ischæmic muscular contracture affecting the small muscles of the hand alone.

The final contracture is due to dense fibrosis, but in the earliest stages there is contracture due, as Volkmann said, to *rigor mortis* of the muscles. Within a very few days, the affected muscle becomes a yellowish-green homogeneous mass, firm to the touch and avascular. If neighbouring muscles are affected they cannot be distinguished separately. Later, the mass acquires a fibrous sheath and contracts. Finally there may be calcification.

Microscopically the changes are those of an infarct surrounded by a zone of living striated muscle nourished by marginal blood-vessels. The longitudinal striations of the muscles are lost, while the transverse striations become more and more accentuated until the muscle fibres become transversely fragmented. Soon there is a central area of necrotic muscle and connective tissue fibres.

An interesting possibility has become apparent recently. Le Gros Clark (1946) has shown that a rabbit can replace its *tibialis anterior* muscle within four months of the arterial blood supply being cut off, the new muscle coming from the surface layer of surviving muscle fibres. In the human it is the greater mass of necrotic muscle which prevents regeneration. The regenerating muscle fibres are guided by the old endomyseal tubes, but fibrosis destroys the more distant part of the endomyseal framework before the muscle fibres reach there. A case of partial regeneration of the human *tibialis anterior* has been reported (Horn and Sevitt, 1951), and it is probable that some recovery occurs in Volkmann's contracture more often than we realize.

The cause of the muscle necrosis has been debated ever since 1872, when Richard von Volkmann realized that it was not due to nerve injury; he himself later suggested ischæmia. The theory that it is caused by venous obstruction was held for many years, but Griffiths (1940) established the basic cause as arterial ischæmia. The appearance of central necrosis and surrounding reaction is that of an arterial infarct; this contrasts both with the diffuse interfibrillary exudate and fibrosis found in venous obstruction, and with the early microscopic picture and the late proliferation of connective tissue septa in nerve lesions. It is not the picture of a partial arterial ischæmia; in this the muscle fibres degenerate, but the intermuscular connective tissue survives. Venous occlusion may well be an aggravating factor.

Except in the mildest cases, one or more nerves are usually affected as well as the muscles. The median nerve is most commonly involved, the ulnar next and the radial least. The cause of the nerve lesions as of the muscular lesions is usually arterial ischæmia, although they may be damaged by the initial injury or even by late fibrotic contracture of muscles (for example, between the heads of the *pronator teres*). The blood supply of the median nerve is from a main artery branching off the anterior interosseous artery and from collateral vessels along the course of the nerve. If only a part of this blood supply is affected, the duration of the lesion may be short and the effects mild. In a severe lesion the effect is complete and permanent; in such a case the nerve is shrivelled and hard, and all the structures within it are replaced by a dense mass of collagen (Holmes, Highet and Seddon, 1945).

The skin is rarely affected in the early stages apart from concomitant pressure sores. Later there may be trophic changes in the skin and nails. Griffiths (1950) has seen ischæmic necrosis of bone in a case of muscular contracture. Secondary bone and joint deformities may appear later.

The Causes of Arterial Obstruction.

The arteries concerned are the brachial and its branches and collaterals near the elbow. The usual lesion is an intense spasm of the brachial artery starting abruptly an inch or two above its bifurction and spreading downwards to involve the radial and ulnar arteries. The branches of all these vessels may also be in spasm; but even if not themselves constricted, they can be effectively occluded at their origin by spasm of the main arteries.

The cause of the arterial spasm cannot yet be regarded as definitely established. It is probably due to direct stimulation of the smooth muscle in the arterial wall by stretching or by mechanical trauma; but it has in the past

been regarded as due to a local periarterial autonomic reflex or to a spinal autonomic reflex.

The chief experimental basis of the spinal reflex theory was the work of Barnes and Trueta (1942) on rabbits. They applied a tight wire tourniquet to one hind limb and found arterial spasm in both hind limbs; it was concluded that this was due to a spinal autonomic reflex. The situation has been clarified by the work of Kinmonth and others (1949 and 1952), who repeated Trueta's work, but used more safeguards and took more precautions. They found that the sizes of the arteries varied with the carotid blood pressure, and that there was no difference between sympathectomized and normal animals. They also found that the rabbit's upper femoral artery responded by spasm to mechanical trauma and to stretching, but did not respond to efferent autonomic stimulation. The rabbit's lower femoral artery responds both to direct mechanical stimulation and to efferent autonomic stimulation. The human lower brachial artery certainly responds to mechanical stimulation and is probably able to respond to efferent autonomic stimulation as well. As far as possible, Kinmonth's rabbit experiments have been attempted during operations on humans, and similar results have been obtained (1952*b*).

While reflex nervous spasm of the human lower brachial artery may occur, it is not likely that it is of practical importance in the ischæmia of early Volkmann's contracture. Spasm of large vessels has been seen repeatedly in operations when the sympathetic paths have been blocked by spinal anæsthesia, by local anæsthesia of the arterial segment concerned, and by interruption of peripheral nerves. In the rabbit, spasm due to nervous stimulation lasts only for the duration of the stimulus, while mechanical spasm may last for hours or days, and may spread up and down the artery into its branches.

The spasm found in Volkmann's ischæmia is probably always the mechanical type. Supracondylar fracture of the humerus with backward displacement of the distal fragment provides a very suitable stimulus. It gives the brachial artery a sudden stretch and can pull the artery against a sharp piece of bone.

Any of the mechanisms affecting the main artery can affect its collaterals, but the latter can be occluded much more easily by external pressure. In their experiments Kinmonth *et alii* recorded the volume of the rabbit's paw before and during mechanical induction of spasm of the femoral artery. The volume of the paw remained virtually unchanged, and from this it was deduced that the collaterals were not obstructed.

In addition to spasm there may be accessory factors causing ischæmia. These are external pressure, low blood pressure and thrombosis.

External pressure may be due to deformity, to pinching by bone fragments, to tight splints, bandages or plasters, and to œdema in rigid fascial compartments. An exudate or hæmatoma can possibly compress small vessels if the elbow is flexed or if the blood pressure falls after their formation. The position of the elbow is of great importance, because flexion not only increases the pressure beneath the deep fascia, but also causes a tight fascial band to press inwards. A similar band can be seen easily if a child's sausage-shaped rubber balloon is bent in the middle.

The possibilities of thrombosis have not received much attention in recent years. Secondary capillary obstruction by "conglutination" of the cells may sometimes determine the fate of muscles, even though the arterial obstruction is relieved (Harman, 1948).

The reason for the extraordinary distribution of the contracture is obscure. Why should the deep flexors of the digits be involved so regularly and the extensors so rarely? Why should the small muscles of the hand not be involved?

The work of Blomfield (1945) on the anatomy of the muscle blood vessels in the leg does suggest a possible reason.

> In the gastrocnemius the vessels are commonly derived from a single main artery which splits up into branches to enter the upper end of the muscle and descend throughout its entire length.

One can imagine that if the most commonly affected muscles were supplied by end arteries arising high in the forearm, then these vessels would be occluded early by a spasm spreading downward from above, while muscles without this type of blood supply could be kept alive by the collateral circulation. Unfortunately for this hypothesis it is not supported by the researches of Salmon and others (1933 and 1935), who have done extensive and detailed work on the arteries of the arm.

Clinical Features.

The patient is usually a child who has sustained an injury of the elbow region or of the forearm. The onset of ischæmia usually occurs a few hours after the injury and often closely follows manipulation of a fracture. Spasm of the artery may last only a few minutes or as much as four days.

A burning pain in the forearm is the commonest symptom, but may be absent if there are extensive nervous lesions. Pain is worse on active or passive movements of the fingers, particularly extension. It may disappear in two days, even though the ischæmia is unrelieved.

The radial and ulnar pulses are usually absent, but may be weakly felt. The forearm and hand are swollen and cold, usually cyanosed but perhaps pale. The fingers begin to flex, often first at the distal interphalangeal joints; this finger flexion indicates structural damage to the muscle. After a few days there are signs of inflammation, which is the body's reaction to the dead tissue and gave rise to Volkmann's original term of "myositis".

Slowly the swelling settles, the hard muscles can be felt and the contracture gradually tightens. The late deformity in a severe case is characteristic. The elbow is flexed and the forearm pronated; the fingers are flexed at the interphalangeal joints when the wrist is extended, but this is lessened when the wrist is flexed. Children often develop a trick finger-flexion grip by extending the wrist. Metacarpo-phalangeal movement is usually quite good unless there is an ulnar nerve palsy, in which case hyperextension is present. There may be the "ape thumb" deformity of a median nerve lesion.

In severe cases there are trophic changes in the skin and nails. The soft tissues, bones and joints may be affected in time, and the whole of the affected arm may eventually be smaller than the normal arm. The median nerve is affected more often than the ulnar. Nerve lesions usually start at the same time as the muscle necrosis, but may ensue as much as a year afterwards. In either case they may recover. As a rule, the area of sensory loss in early cases cannot be recognized as typical of any nerve.

Diagnosis is easy in a typical case. In early and doubtful cases the radial pulse is the best guide, but is not infallible. If available, an oscillometer is a help. Griffiths (1953) has suggested that if a water-soluble radio-opaque solution was injected into the suspected area and this was examined radiologically about half an hour later, it would help the diagnosis in doubtful cases. In ischæmic lesions the solution would remain present for a much longer period. However, no work has yet been done to distinguish between the absorption times in ischæmic muscles and in muscles which are only congested by venous obstruction. Griffiths has also suggested that injection of a muscle relaxant might distinguish between very early ischæmic contraction and reflex contraction of the finger flexors.

Treatment.

Prevention depends on remembering the possibility of ischæmia and on manipulating elbow and forearm fractures in children very gently. All patients with severe fractures of this nature should be admitted to hospital until the danger has passed, so that a frequent check can be made of the pulse, colour, temperature, sensation and blood pressure.

The fracture should be gently reduced. Traction during reduction should be minimal, because stretching is an excellent stimulus to spasm. Above all, the technique of exaggerating the initial deformity should be avoided as much as possible. Useful as this manœuvre may be, it stretches over a jagged piece of bone a sensitive artery which is already taut. When the fracture is reduced the elbow should be immobilized in a position some 10° short of the position in which the circulation starts to be impaired. If used at all, splints, bandages and plaster casts must allow room for swelling. Elevation minimizes swelling, and injection of hyaluronidase may help to dissipate it (Gartner and MacAusland, 1954).

If ischæmia is apparent, it must be relieved within six hours of its onset if contracture is to be avoided.

Shock or low blood pressure must be treated; the fracture must be reduced; flexion of the elbow should be as little as is compatible with maintaining reduction of the fracture; and all constricting appliances should be removed. The limb should be kept cool to reduce its metabolism. If shock allows, heating the rest of the body to 110° to 115° F. dilates the collaterals as long as they are not occluded by external pressure or by mechanically induced spasm; but even so, it is doubtful whether the blood supply of the affected muscles is improved.

Sympathetic blocks in the paravertebral and cervical plexus regions will remove the normal vasoconstrictor tone of the collaterals. This may or may not improve the blood supply of the forearm as a whole, but it probably has no effect on the ischæmic muscles. Dramatic effects have been ascribed to these sympathetic blocks, but their value has always been strongly contested; apparent benefits have been ascribed to coincidence and to cutaneous vasodilatation.

Open operation and release of pressure have long been practised, and success has been claimed for them. After the deep fascia has been incised, it is often difficult to close the skin wound; therefore many short skin incisions in the same line may be used to divide the fascia extensively.

Periarterial sympathectomy and arterectomy used to be performed on arteries which were in spasm although otherwise healthy. If part of an artery is so badly damaged that thrombosis is occurring, excision is indicated.

Kinmonth (1952*a*) has recommended open operation and the application of smooth-muscle relaxants to the artery if it is in spasm. In his experiments on traumatic spasm of the rabbit's femoral artery, he found that the only measure which constantly relieved the spasm was the local application of smooth-muscle relaxants. The same agents were ineffective when given intravenously, because spasm of the arterial wall occluded the *vasa vasorum*. The most effective agent was 2·5% papaverine sulphate solution, which induced immediate, complete and permanent relaxation, while procaine, "Priscol" and caffeine had temporary effects. Pethidine has been shown to be as effective as papaverine, but is much more toxic (Kinmonth, 1952*b*).

Papaverine is one of the many drugs found in opium, but it is not related chemically or pharmacologically to morphine. It is a smooth-muscle relaxant, and large doses have little toxic effect. In a small series of patients, Kinmonth (1952) has demonstrated that its effect on the human arteries is the same as on those of the rabbit. Kinmonth advises open operation because it is the only way to find out whether the occlusion is due to spasm or to organic obstruction; and in either case it is only by operation that effective treatment can be applied.

For the established contracture, several lines of treatment are possible. Conservative treatment by splinting is effective for mild contractures and should precede operative procedures even in severe cases. Splinting should be started as early as possible to prevent the contracture from developing; worthy an opponent as the fibroblast may be, it is not as formidable as the fibre it lays down. The best technique is that of Robert Jones, in which the individual fingers are first splinted in extension while the wrist is flexed, and then the wrist is extended on another splint. The splints are removed for active exercises three or more times a day. In most cases improvement can be effected in six to twelve months, but in only the mildest cases will the patient be left with a good hand.

The results of forced manipulation under anæsthesia are bad.

If it is the superficial muscles which are most affected, early excision is straightforward. If, as is usually the case, the deep muscles are most affected, it is difficult to excise the fibrotic mass without damaging the remaining healthy muscles or their neurovascular supply. Especially in the early stages, it is difficult to decide clinically exactly which muscles are affected; but perhaps this problem will be eased by the use of curare or radioactive isotopes.

One of the early operative treatments was Max Page's operation (1923), in which the flexor origins at the medial epicondyle were detached and allowed to slide distally. Page did not design the operation specifically for Volkmann's contracture, but for all forearm flexion contractures; in his original series there was only one case of Volkmann's contracture. Best results are again likely in those unusual cases in which the superficial muscles are most affected.

Garré shortened the forearm bones by resection of three-quarters to one inch from each bone. This may be followed by non-union due to trophic changes in the arm. Hemicarpectomy and total carpectomy are similar in principle, in that they shorten the forearm; or all the tendons may be lengthened.

All the foregoing procedures attempt either to shorten the bow or to lengthen the bowstring; they accept the artificial tenodesis and try to lengthen it. However, some tendon transfers have proved effective. The tendon of one of the extensors may be transferred to the finger flexors with arthrodesis of the wrist. Again, if the *flexor sublimis* muscle is working and the *flexor profundus* contracted, it is possible to divide the sublimis tendons distally and the profundus tendons proximally, and then to attach the proximal ends of the sublimis tendons to the distal ends of the profundus tendons. Parkes (1951) has introduced a new concept by taking advantage of the trick finger-flexion grip which children acquire by wrist dorsiflexion. After tenotomizing the flexor tendons he detaches the insertion of the *extensor carpi radialis longus* and button-holes it to the tendons of the *flexor digitorum profundus*. He divides the tendon of the *flexor pollicis longus* to permit extension of the thumb, but he does not include it in the tendon transfer because there is little disability as long as the thumb can be opposed, and because if it was included finger flexion and thumb flexion could not be performed separately. The only disability is that the child cannot pinch with the thumb nail, as in picking up a threepenny piece from a hard table top (Parkes, 1952). Parkes attributes his bad results to subclinical involvement of the *extensor carpi radialis longus* by the ischæmia, and to damage to the wrist joint at the time of the original injury. Parkes regards his operation, not as a transplantation of an extensor to do the work of a flexor, but as a supplement to finger flexion by a muscle which is accustomed to work in synergy with the finger flexors.

Prognosis.

In most cases acute ischæmia can be relieved by conservative measures, and no contracture results.

If there is an acute flexion deformity of the fingers, or if there is pain in the forearm on active or passive extension of the fingers, irreparable damage has already been done, although this may not necessarily be enough to cause gross contracture. If ischæmia exists for six hours or more, contracture is probable. However, definite cases of spasm are sometimes not followed by contracture; perhaps in these cases the spasm does not spread low enough to obstruct the arteries to the muscles. On the other hand, it is possible that shorter periods of ischæmia may lead to irreparable capillary obstruction, and that contracture may result even if the circulation is relieved as soon as three hours after its beginning. In the first few months, contracture increases and no certain prognosis can be given.

With a cooperative patient, intelligent parents and an early start, persistent conservative methods may give surprisingly good results; this may perhaps be partly due to regeneration of muscle fibres. In late cases there is no recovery. It is well to remember that patients with severe untreated contractures are able to undertake heavy work, and I have seen one such, with an associated median nerve lesion, who had worked as a dock labourer in Liverpool for thirty-seven years. Adjusting the lengths of bones and tendons may improve the appearance, and to a less extent the function. In suitable cases Parkes's operation can give excellent results and other tendon transfers good results.

References.

BARNES, J. M., and TRUETA, J. (1942), "Arterial Spasm, an Experimental Study", *Brit. J. Surg.*, 30: 74.

BLOMFIELD, L. B. (1945), "Intramuscular Vascular Patterns in Man", *Proc. Roy. Soc. Med.*, 38: 617.

CLARK, W. LE GROS (1946), "An Experimental Study of the Regeneration of Mammalian Striped Muscle", *J. Anat.*, 80: 24.

GARTLAND, J. J., and MACAUSLAND, W. R., junior (1954), "Use of Hyaluronidase in Soft Tissue Injury and Its Influence on Experimental Bone Repair", *Arch. Surg.*, 68: 305.

GRIFFITHS, D. L. (1940), "Volkmann's Ischæmic Contracture", *Brit. J. Surg.*, 28: 239.

GRIFFITHS, D. L. (1950), "Certain Vascular Lesions", in "Modern Trends in Orthopædic Surgery", Butterworth, London.

GRIFFITHS, D. L. (1953), Personal communication.

HARMAN, J. W. (1948), "The Significance of Local Vascular Phenomena in the Production of Ischæmic Necrosis in Skeletal Muscle", *Am. J. Path.*, 24: 625.

HOLMES, W., HIGHET, W. B., and SEDDON, H. J. (1945), "Ischæmic Nerve Lesions Occurring in Volkmann's Contracture", *Brit. J. Surg.*, 32: 259.

HORN, J. S., and SEVITT, S. (1951), "Ischæmic Necrosis and Regeneration of the Tibialis Anterior Muscle after Rupture of the Popliteal Artery", *J. Bone & Joint Surg.*, 33B: 348.

KINMONTH, J. B., SIMEONE, F. A., and PERLOW, V. (1949), "Factors Affecting the Diameter of Large Arteries with Particular Reference to Traumatic Spasm", *Surgery*, 26: 452.

KINMONTH, J. B. (1952a), "The Physiology and Relief of Traumatic Arterial Spasm", *Brit. M. J.*, 1: 59.

KINMONTH, J. B. (1952b), Personal communication.

KINMONTH, J. B., and SIMEONE, F. A. (1952), "Motor Innervation of Large Arteries with Particular Reference to the Lower Limb", *Brit. J. Surg.*, 39: 333.

PAGE, C. M. (1923), "An Operation for the Relief of Flexion-Contracture in the Forearm", *J. Bone & Joint Surg.*, 5: 233.

PARKES, A. (1951), "The Treatment of Established Volkmann's Contracture by Tendon Transplantation", *J. Bone & Joint Surg.*, 33B: 359.

PARKES, A. (1952), Personal communication.

SALMON, M. (1933), *"Les artères des muscles des membres et du tronc"*, Masson, Paris, 39.

SALMON, DOR, LÉNA, A. (1935), *"A propos la pathogénie de la maladie de Volkmann. Recherches sur les artères des muscles de l'avant bras"*, *Bull. et mém. Soc. de chir. de Marseille*, 7: number 4.

PREVENTION OF HYALINE MEMBRANE AFTER CÆSAREAN SECTION.

By JUNE PASH, M.B., B.S.,

Medical Superintendent, Queen Victoria Memorial Hospital, Melbourne.

HYALINE MEMBRANE is one of the major causes of mortality in premature infants and also in mature infants delivered by Cæsarean section. The Boston theory is that hyaline membrane is the result of aspiration of amniotic fluid into the respiratory tract and the precipitation of the protein content in the alveolar ducts to form a membrane. If we accept this, one might expect that a baby delivered by Cæsarean section, particularly if the operation was elective and the membranes were intact at the time of operation, would be in great danger of inhaling this fluid. To this is added the fact that with modern anæsthetic methods the baby quite frequently breathes while the head is being delivered through the incision. Under such circumstances, then, the chances of inhalation of liquor are great. The same principle holds in vaginal deliveries in cases of hydramnios, in which the membranes rupture late, and in delivery of the second twin, in which the membranes are often ruptured just before delivery of the child.

After consideration of these facts it seemed obvious that some attempt should be made to suck out the respiratory passages of the baby delivered by Cæsarean section before this aspiration could take place, and the following method has been used.

An assistant, who is present at the operation purely for the purpose of resuscitating the baby, is scrubbed, gowned and gloved. The metal sucker is replaced by a number 8 rubber catheter connected to the heavy suction tubing by a metal connexion. This is tested before the operation is commenced. After the incision in the lower segment is made, blood and liquor are sucked away and the excess is removed by swabbing with packs if necessary. If the head is low enough, the baby is usually delivered by the face, and in this case the assistant, as soon as the mouth is accessible, places the catheter in the mouth, and mouth and pharynx are cleaned before the child's head is delivered. This suction is maintained by the assistant throughout the delivery of the head, which is usually done with forceps. Sucking out of the mouth and pharynx is continued while the child's body is delivered, and then the child is held with its head down. As soon as the cord is clamped and cut, the assistant, carrying the child with the head low, takes it to a cot, which is inclined with head down at an angle of 30°, and the stomach is then emptied of ingested liquor with a mucus catheter, and further resuscitation measures are carried out if required.

If the head is high it may be difficult to gain access to the mouth, and in these cases suction is instituted by the assistant as soon as the mouth is accessible, and delivery of the child is delayed until the airway is reasonably clear. In those cases in which the baby is delivered by the breech, suction is again commenced as soon as the mouth is accessible, and the head delivered slowly while suction is continued.

Gellis recommended that babies delivered by Cæsarean section should have their stomach contents aspirated. We have used this method for some time in resuscitating babies delivered by Cæsarean section, but have found that this alone was insufficient to eliminate the occurrence of hyaline membrane.

We have been using the reported method at this hospital for twelve months, and in 60 elective Cæsarean sections have had no deaths from hyaline membrane. We have also reduced neonatal morbidity from atelectasis in these cases.

Acknowledgement.

I wish to thank the members of the obstetric staff of the Queen Victoria Memorial Hospital, Melbourne, for their cooperation in the use of this method.

THE NEURO-PATHOLOGY OF THE LANDRY-GUILLAIN-BARRÉ SYNDROME.

By R. D. K. REYE,

From the Department of Pathology, Royal Alexandra Hospital for Children, Sydney.

THIS study is based on a group of six fatal cases with autopsy, in which the clinical diagnosis at the time of death was acute anterior poliomyelitis. These cases occurred during a period extending from December, 1949, to April, 1951, a period during which cases of poliomyelitis were occurring in the community with considerable frequency.

The reason for this brief report is simply to demonstrate, firstly, the necessity for a full pathological examination of the peripheral as well as the central nervous system of all patients who have died of respiratory paralysis, more

especially during a poliomyelitis epidemic, at which time the correct clinical diagnosis may be obscured. Secondly, the report is written to put on record a further series in this ætiologically ill-defined syndrome—a series in which the morbid anatomical changes can be shown to exist in all the cases examined; there are many similar cases in the literature, to which the name of this syndrome is attached, in which no significant pathological changes have been demonstrated.

The age and sex of the patients and the duration of the fatal illness are set out in Table I. The first column contains the record number from the autopsy file, and this number will be used for reference purposes in the text.

TABLE I.

Serial Number.	Age.	Sex.	Duration of Illness. (Days.)
5259	5·5 years.	M.	13
5475	3 years.	F.	6
5507	11 years.	M.	34
5989	5 years.	M.	8
6149	5 months.	M.	4
6257	4 years.	M.	6

In each case death occurred as the result of paralysis of the respiratory muscles. The disease progressed rapidly to a fatal issue within fourteen days in all cases, with one exception. This patient was the oldest in the series, and in this case the illness lasted for just over a month before terminating fatally.

Pathological Investigation.

Central Nervous System and Lepto-Meninges.

Gross examination of the brain and spinal cord and its covering meninges revealed nothing of diagnostic significance. The brain tissue was moderately œdematous in all cases, and there was "lipping" of the edges when transverse cuts were made across the spinal cord. The cerebral and spinal vessels and the vessels of the meninges were engorged with blood, but a comparable degree of congestion is found very often in quite unrelated conditions. Petechial hæmorrhages were present in one case only (6149), with a distribution restricted to the grey matter of the spinal cord.

No pathological changes were found in the brain on microscopic examination, and the cerebral meninges were normal except in one case (5259). In this case there was a slight and patchy inflammatory reaction, indicated by an increase in size and number of the fibrous tissue cells and a small accumulation of lymphocytes and larger mononuclear cells. Cellular infiltration of the lepto-meninges of the spinal cord, though present to a greater degree, was also relatively insignificant. Small patchy infiltrations, mainly perivascular in position, were present in three cases (5259, 5989, 5475), and none of these were more extensive than the focus illustrated in Figure I.

In contrast to the cerebrum and the cerebellum, microscopic examination of the spinal cord and brain stem did reveal pathological changes in all but one case (6149), and of these changes the most striking, and the only one evident in sections stained by hæmatoxylin and eosin, was a moderate to severe chromatolysis in the motor neurons of the spinal cord and other neurons of the peripheral nerves. This degenerative change was especially marked in Case 5507, in which vacuolation of the now pallid cytoplasm was a pronounced feature. No areas of demyelination were found in sections stained to demonstrate the myelin sheaths. In two cases (5259, 5475) slight swelling and irregularity in outline of the axis cylinders were seen in the silver impregnated material. These axonal changes were present in the anterior commissures of the grey matter and in a few isolated fibres in the white matter.

Spinal and Peripheral Nerves.

Pathological alterations in varying degree were present in the nerve roots of the spinal cord in all cases, being least advanced in Case 6149, the only case in the series in which no chromatolysis of the neurons had occurred. In all cases the lesions were irregular in their distribution and involved only a portion of a nerve fibre or a segment of its length. Except in the earliest stage it was possible to gain some insight into the essential pathology from the preparations stained by hæmatoxylin and eosin; but special staining techniques, especially for the axis cylinders, were necessary for a full appreciation of the damage to which the nerve fibres had been subjected and to appreciate slight and early structural changes. Œdema, with separation of the constituent elements of the nerve fibre, was a pronounced feature in some of the cases, and this œdematous change is present to a marked degree in the anterior nerve root illustrated in Figure II; but it was not present in all nerves in that case, nor to the same degree in all cases, and was sometimes found in nerves showing no other evidence of damage.

By silver impregnation methods, the extent and degree of degeneration of the axis cylinders were rendered very obvious. In the early stages this took the form of an irregular swelling and beading of the axis cylinders; changes of that type were present in Case 6149. This case had an approximate duration of four days, and was the only case in the series in which no chromatolysis of the neurons was found. This is consistent with the belief that these changes in the neurons are the result of retrograde degeneration following upon axonal damage. The degree of axonal degeneration was only slightly more marked in Case 6257, though reference to Figure III reveals definite slight swelling and distortion of some of the axis cylinders. To judge from the clinical story, the duration of the disease in this case was less than a week, though definite chromatolysis was present in the motor neurons. Very considerable axonal degeneration was present in the spinal nerves in the remaining four cases, principally in the anterior roots, though posterior roots were not exempt. In these cases the axonal swelling and irregularity reached a considerable degree (Figure IV), and there was fragmentation of the axons extending to almost total disappearance of the fibres in the case of longest duration (5507); this is illustrated in Figure V.

Wherever axonal degeneration occurs, proliferation of the neurolemma nuclei is likely to follow, and the greater the duration of the disease, the more likely is this hyperplasia to be encountered. Haymaker and Kenohan (1949), on the basis of their study, suggest that this increase of the neurolemma cells is most likely to be found after the thirteenth day from the onset of the illness, though they described one case in which this change was found as early as the ninth day. It is interesting therefore to find, in this series, that by far the greatest proliferation of the neurolemmal cells was to be found in Case 5507, with an estimated duration of thirty-four days (see Figure VI). Slight to moderate proliferation of these cells was present in two additional cases in which the duration of the illness was six to thirteen days respectively, but was absent in the remaining three cases. Lymphocytes and macrophages, usually considered as evidence of the beginnings of resolution and reparative changes in lesions of the type found in these cases, were present in the spinal nerves in one case only (5507), the case of longest duration in this group. Lymphoid cells can be recognized in Figure VII as small, darkly staining nuclei, in contrast to the large, pale nuclei of the cells of Schwann, which have undergone multiplication, while in Figure VIII, a portion from Figure VII which has been more highly magnified, the macrophages, some with foamy cytoplasm, can be seen almost to fill a space formerly occupied by a myelinated nerve fibre.

Tissues other than Neural.

Evidence of inflammation of the upper part of the respiratory tract was present in three cases; this was of mild degree in one, but severe in two, with extensive

necrosis and membrane formation in one of these. Needless to say, the possibility of diphtheritic infections had been rigidly excluded in all cases, and a *Staphylococcus aureus* was the predominating organism obtained from cultures taken from the trachea at autopsy. In two of the cases in which laryngeal and tracheal inflammation was present, bronchopneumonia was an additional finding, and bronchopneumonia was also present in one other case in which the upper part of the respiratory tract was not inflamed. In two cases there was no gross abnormality.

Histological examination of all organs was not made in every case. Sections of the heart, liver and kidneys were available in all cases, and in none of these were any pathological changes to be found. In sections of the adrenals, pancreas, thyroid and pituitary, which were taken in some cases, no abnormality was detected. The spleen and lymph nodes were enlarged in two cases. In one of these there was hyperplasia of the pulp cells, in the other the pulp was packed with polymorphonuclear leucocytes. In the lymph nodes non-specific hyperplasia was evident.

Discussion.

Opportunity for pathological study of subjects of this syndrome does not occur very frequently. In only a small number of sporadic cases have the patients come to autopsy in this hospital prior to the group reported in this instance, and only two patients since. Much confusion still exists regarding the exact pathology, and to some extent this arises because there are many reports in the literature dealing with examples of this syndrome, as judged by clinical criteria, in which no pathological changes have been found, and such reports colour the descriptions recorded in many standard works of reference. It is accepted that in those examples in which there is rapid progression passing to a fatal issue in under four days, pathological alterations, apart from non-specific œdematous swelling, are unlikely to be demonstrable. Whether similar cases of longer duration can also occur without detectable pathological alteration in the nerve roots remains a matter for speculation. It is important that this point should be clarified as far as possible, and this can best be done by careful investigation and reporting of fatal cases with the use of selective staining techniques. It cannot be too strongly emphasized that pathological reports, based solely on observations made on hæmatoxylin and eosin sections, as has occurred not infrequently, are likely to be inadequate and therefore misleading. Haymaker and Kenohan (1949) have done much to clarify the position in their comprehensive article, which contains a carefully analysed survey of the literature and a report of 50 fatal cases thoroughly studied, and their report serves to place the pathology of this condition on a sound basis. Scheinker (1949), in his report of 10 fatal cases, presents pathological features which, except for changes in the cord itself, do not differ materially from those of the last-mentioned authors, and he considers the changes which he describes as being quite characteristic.

Summary.

The pathology, as expressed in six fatal cases of the Landry-Guillain-Barré syndrome, is described. Emphasis is placed on the need to examine, by the appropriate histological techniques, the peripheral nervous system in any cases suspected of belonging in this syndrome.

References.

HAYMAKER, W., and KENOHAN, J. W. (1949), "The Landry-Guillain-Barré Syndrome. Clinicopathologic Report of Fifty Fatal Cases and a Critique of the Literature", *Medicine*, 28 : 59.

SCHEINKER, I. M. (1949), "Pathology and Pathogenesis of Infectious Polyneuritis (Guillain-Barré Syndrome)", *J. Neuropath. & Exper. Neurol.*, 8 : 184.

Legends to Illustrations.

FIGURE I.—Perivascular accumulation of lymphoid cells in the leptomeninges of the spinal cord (hæmatoxylin and eosin stain, x 200).

FIGURE II.—Separation of the constituent fibres of an anterior nerve root by œdema fluid (hæmatoxylin and eosin stain, x 200).

FIGURE III.—Anterior nerve root from Case 6257. Slight swelling and distortion of the axis cylinders (silver impregnation, *Roger's method, x 200*).

FIGURE IV.—Considerable swelling and distortion of the axis cylinders in an anterior nerve root (silver impregnation, *Roger's method, x 450*).

FIGURE V.—Anterior nerve root from Case 5507. Fragmentation and disappearance of axons (silver impregnation, Roger's *method, x 200*).

FIGURE VI.—Anterior nerve root from Case 5507 in cross-section. The abundance of darkly staining nuclear material is due to a proliferation of the neurolemmal cells (hæmatoxylin and eosin stain, x 200).

FIGURE VII.—Alterations indicative of resolution and repair from an anterior nerve root, Case 5507 (hæmatoxylin and eosin stain, x 200).

FIGURE VIII.—Portion of Figure VII to show macrophages, some of which are foamy, replacing a disintegrated axis cylinder (hæmatoxylin and eosin stain, x 450).

Reports of Cases.

PATENT DUCTUS ARTERIOSUS WITH ATYPICAL SIGNS.[1]

By J. M. GARDINER,[2]
Alfred Hospital, Melbourne.

THE classical signs of patent *ductus arteriosus* are well known (Taussig, 1947; Gross and Longino, 1951). The most important is the continuous or machinery murmur (Gibson, 1900); without it the diagnosis becomes difficult and uncertain. That the typical murmur may not be present in infants and young children is well recognized. What is not so generally known is that under certain circumstances a patent *ductus arteriosus* may be present in older children and in adults in the absence of this murmur. Moreover, when this occurs it is usually due to the presence either of a very large ductus, or of pronounced pulmonary hypertension, or of both. As the prognosis in these circumstances is worse than in the ordinary case of patent *ductus arteriosus* with typical murmur, which is readily diagnosed and presented to the thoracic surgeon for ligation, the recognition of these cases is especially important. Their clinical features may closely resemble those of ventricular or of atrial septal defects, and certain differentiation may be possible only by cardiac catheterization.

This paper presents three illustrative cases which have been investigated at the Alfred Hospital in the past eighteen months. One is an example of a very large ductus; the other two patients had severe pulmonary hypertension. In one of these two cases this had not yet caused significant reversal of flow through the ductus; there was no clinical cyanosis, and operation was undertaken successfully. In the other the pulmonary artery pressure equalled that in the aorta and there was reversal of flow through the ductus with clinical cyanosis; operation at this stage was considered inadvisable. All three patients had a history of maternal rubella in the first trimester of pregnancy.

Case I.

A girl, aged twelve years, had had a heart murmur discovered in infancy. She had always been small for her age, and subject to recurrent attacks of pneumonia. She was easily tired, and became short of breath on moderate exertion; she had never been cyanosed. She had been under observation for some years, and regarded as suffering from Lutembacher's syndrome. Her mother had suffered from rubella when two months pregnant.

Examination showed her to be a small girl for her age; her weight was four stone. She was acyanotic and there

[1] Two of the cases reported were presented at a meeting of the Melbourne Paediatric Society on September 9, 1953.

[2] Portion of this work was done during the tenure of the Victor Y. and Margaret Kimpton Research Fellowship, 1953.

was no clubbing of fingers or toes. Her radial pulse had a striking waterhammer quality, and her blood pressure was 135 millimetres of mercury, systolic, and 0 millimetre, diastolic. The femoral pulses were of the same quality and not delayed. There was a slight left-sided precordial bulge. Her cardiac impulse was a strong left ventricular thrust; she had a slight lift over the right ventricle, but no palpable pulmonary artery pulsation. The second heart sound at the pulmonary area was narrowly split and accentuated. At the apex there was a loud blowing systolic

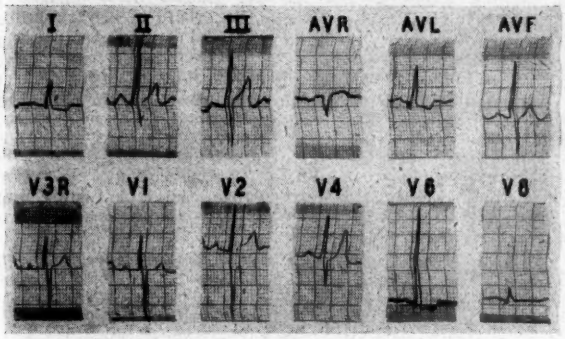


FIGURE I.
Electrocardiogram from Case I, showing left ventricular preponderance. All precordial leads are at half-normal sensitivity.

murmur and a loud mid-diastolic murmur accompanied by a well-marked thrill. A blowing early diastolic murmur was heard to the left of the sternum in the second and third left intercostal spaces. No continuous murmur was heard anteriorly, but when the result of catheterization was known, on careful auscultation a faint suggestive murmur was heard at the angle of the left scapula.

The electrocardiogram (Figure I) showed a *P mitrale*, and the pattern of left ventricular preponderance. X-ray

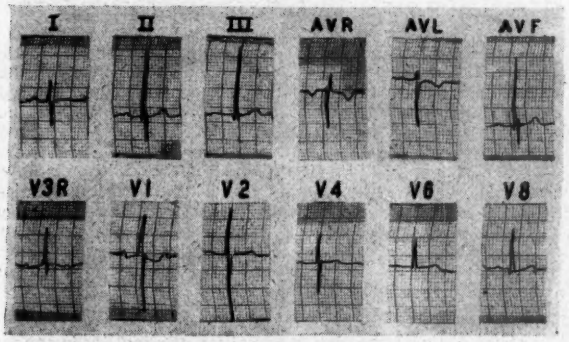


FIGURE II.
Electrocardiogram from Case II, showing slight right ventricular dominance. All precordial leads are at half-normal sensitivity except V_3R, V_1 and V_8

examination (Figure IV) showed the heart to be large, and pulmonary plethora was present. Fluoroscopic screening showed that there was enlargement of the left ventricle, the left auricle and possibly the right ventricle.

Cardiac catheterization was performed on September 5, 1952. The results are summarized in Table I. They revealed a considerable aorto-pulmonary shunt, with moderate elevation of pulmonary artery pressure, though not to more than 60% of the systemic level. Arterial oxygen saturation was normal.

In view of these findings, thoracotomy was advised, and performed by Dr. C. J. Officer Brown on September 16. A very large patent *ductus arteriosus* was found, the largest in the surgeon's series of over 130 cases. The dissection was difficult, and eventually the ductus was ligated with three heavy ties.

The child recovered well from the operation. Before she left hospital, and on follow-up examination at three months, it was noted that, although cardiac enlargement and the apical systolic murmur persisted, the pulse and blood pressure were reduced to normal, and the apical diastolic murmur and thrill had disappeared, as had the early diastolic murmur down the left sternal edge. X-ray examination revealed no significant change in silhouette, but definite diminution of pulmonary plethora. However, six months after operation it was found that, although the pulse remained normal and the blood pressure was 110 millimetres of mercury, systolic, and 70 millimetres, diastolic, the mid-diastolic murmur was again audible at the apex, although short and unaccompanied by a thrill; there was now heard a classical continuous murmur in the left first and second intercostal spaces. This was taken

TABLE I.
Findings at Cardiac Catheterization in Case I.[1]

Site.	Blood Oxygen.		Blood Pressure. (Millimetres of Mercury.)	
	Content. (Volume *per Centum.*)	Saturation Percentage.	Systolic/ Diastolic.	Mean.
Superior *vena cava* ..	8·7	65	—	—
Right auricle	8·1	61	—	0
Right ventricle (body) ..	8·3	62	—	36
Right ventricle (high) ..	8·4	63	—	—
Pulmonary artery (main) ..	11·4	85	78/50	64
Pulmonary artery (right) ..	11·2	84	—	—
Femoral artery	12·7	95	135/0 (cuff)	—

[1] Blood oxygen capacity, 13·3 volumes *per centum*; systemic blood flow, 2·6 litres per minute; pulmonary blood flow, 7·3 litres per minute.

as evidence of recanalization; the child has since undergone a second thoracotomy, at which the ductus was successfully divided. At operation the ductus channel was found to be quite small, and the pulmonary artery, which at the previous operation had been grossly dilated, was now almost normal in size.

In the year after the first operation the child grew five inches and gained one and a half stone in weight. Her exercise tolerance was much improved, and she went through the winter without suffering the usual respiratory infections.

Case II.

The patient was a boy, aged fourteen years, whose mother had suffered from rubella when six weeks pregnant. He had always been undersized and thin. He had been able to play some games at school, but could not keep up with other children because of shortness of breath and easy fatigue. No definite cyanosis had been noted.

Examination showed him to be a thin boy with a pigeon chest. He was acyanotic, and showed no clubbing of fingers or toes. His radial pulse had a suggestion of waterhammer quality, but this was not definite, and his blood pressure was 105 millimetres of mercury, systolic, and 65 millimetres, diastolic. His cardiac impulse was a normal left ventricular thrust. There was a slight lifting impulse over the right ventricle, but no pulmonary artery pulsation. The second heart sound in the pulmonary area was narrowly split and accentuated. There was a blowing systolic murmur along the left sternal edge, maximal at the pulmonary area; a short blowing early diastolic murmur was heard localized to the pulmonary area. There was a mid-diastolic murmur at the apex. No continuous murmur was heard.

The electrocardiogram (Figure II) was thought to show evidence of slight right ventricular dominance. X-ray examination (Figure V) showed minimal cardiac enlargement, with a prominent main pulmonary artery and slight pulmonary plethora. Fluoroscopic screening suggested slight right ventricular enlargement and demonstrated increased pulsation in the hilar vessels.

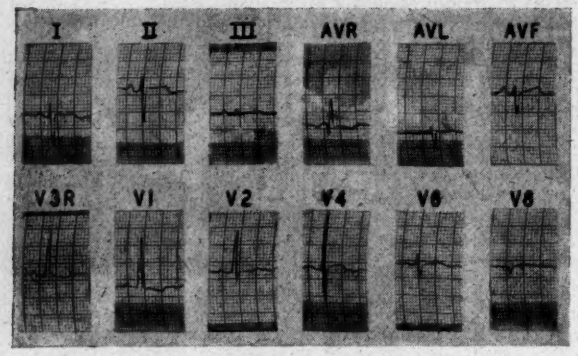


FIGURE III.
Electrocardiogram from Case III, showing right ventricular hypertrophy. All precordial leads are at half-normal sensitivity.

Cardiac catheterization was performed on April 10, 1953. The results are summarized in Table II. They showed a severe degree of pulmonary hypertension, the pulmonary artery pressure being not far below the systemic. There was a rise in oxygen saturation in the pulmonary artery, with an intermediate rise just below the pulmonary valves. The findings were thought best interpreted as those of patent *ductus arteriosus* and pulmonary incompetence. The arterial oxygen saturation was a little below normal, which suggested that in addition to the left-to-right shunt across the ductus there might be a small flow in the reverse direction.

TABLE II.
Findings at Cardiac Catheterization in Case II.[1]

Site.	Blood Oxygen.		Blood Pressure. (Millimetres of Mercury.)	
	Content. (Volumes *per Centum.*)	Saturation Percentage.	Systolic/ Diastolic.	Mean.
Superior *vena cava* ..	12·6	71	—	—
Right auricle	12·6	71	6/0	3·5
Right ventricle (body) ..	12·8	72	90/0	40·5
Right ventricle (high) ..	14·1	80	—	—
Pulmonary artery (main) ..	15·6	88	90/55	72
Pulmonary artery (right) ..	14·7	83	—	—
Pulmonary artery (left) ..	14·8	84	—	—
Femoral artery	16·2	92	105/65 (cuff)	—

[1] Blood oxygen capacity, 17·7 volumes *per centum*; systemic blood flow, 2·8 litres per minute; pulmonary blood flow, 5·0 litres per minute.

In an attempt to localize the site of shunt more exactly, retrograde aortography was attempted on April 14, but was unsuccessful for technical reasons. Accordingly on April 21 Dr. Officer Brown performed a thoracotomy, and found a moderate sized patent *ductus arteriosus*, which was divided.

Subsequently the boy has done well. X-ray examination shortly after operation (Figure VI) showed diminution in heart size, especially in the size of the main pulmonary artery. Clinically the systolic and diastolic murmurs have disappeared. He has gained weight, and has shown improvement in his exercise tolerance.

Case III.

The patient was a girl, aged eleven years, whose mother had suffered from rubella early in her pregnancy. She had been born with defective hearing. From infancy she had had a mild degree of cyanosis, which had not increased in intensity. Her exercise tolerance was good, but she tired easily and could not quite keep up with other children.

On examination, she showed central cyanosis which was slight and variable. There was no detectable difference in colour between the upper and lower extremities. Slight clubbing of fingers and toes was present. Her pulse was normal in quality and her blood pressure was 90 millimetres of mercury, systolic, and 50 millimetres, diastolic. She had a normal cardiac impulse. The second heart sound at the pulmonary area was loud and normally split. There was a harsh pulmonary systolic murmur unaccompanied by a thrill. The electrocardiogram (Figure III) showed the pattern of right ventricular hypertrophy. X-ray examination (Figure VII) showed slight cardiac enlargement, with a prominent main pulmonary artery, but peripheral pulmonary vessels within normal limits, although they were thought to show some abnormal pulsation on fluoroscopic screening.

Cardiac catheterization was performed on August 22, 1952. The findings are summarized in Table III. The catheter was passed from the main pulmonary artery backwards and upwards into the descending part of the aortic arch. A pressure tracing recorded on withdrawal of the catheter from the aorta to the pulmonary artery showed almost identical pressures in both vessels, although the form of the pulse was slightly different. Samples suggested that there was a left-to-right shunt as well as a relatively small right-to-left shunt at the time of examination. However, as it was clear clinically that there was a definite right-to-left shunt for most of the time, which had been present from early life, it was decided that operation should not be performed. When examined a year later, the girl showed no deterioration in her condition or increase in cyanosis.

TABLE III.
Findings at Cardiac Catheterization in Case III.[1]

Site.	Blood Oxygen.		Blood Pressure. (Millimetres of Mercury.)	
	Content. (Volumes *per Centum.*)	Saturation Percentage.	Systolic/ Diastolic.	Mean.
Superior *vena cava* ..	11·5	63·0	—	—
Right auricle	13·1	72·0	—	0·5
Right ventricle (body) ..	13·3	73·0	100/5	54·0
Right ventricle (high) ..	13·2	72·0	—	—
Pulmonary artery (main) ..	16·7	92·0	100/65	87·0
Pulmonary artery (right) ..	14·5	80·0	—	—
Aorta	17·0	93·5	100/65	84·0

[1] Blood oxygen capacity, 18·3 volumes *per centum*; systemic blood flow, 2·6 litres per minute; pulmonary blood flow, 3·4 litres per minute.

Discussion.

It is noteworthy that these patients had been under medical observation for some years. No continuous murmur had been audible, and consequently the diagnosis had remained in doubt. To establish the diagnosis of patent *ductus arteriosus* was especially important in the two acyanotic patients, in whom closure of the ductus was indicated.

The circumstances under which a continuous murmur may not be heard in simple patency of the *ductus arteriosus* are several. First, as was previously mentioned, in infants a systolic murmur only may be audible up to the age of two or three years or even later. It is thought that this is due to the relatively small pressure gradient in the infant between aorta and pulmonary artery, flow through the defect taking place only in systole. It is not until aortic and pulmonary diastolic pressures diverge sufficiently that flow takes place also in diastole (Taussig, 1947).

Secondly, Gross and Longino (1951) mention that in a few proven cases of small ductus there has been heard a systolic murmur only. The lesion in these cases must be very hard to diagnose, and in any case, apart from the accident of infection, it is functionally unimportant.

Thirdly, when a very large ductus is present, as represented by the first case, no typical murmur may be heard. This does not depend on pulmonary hypertension, for the pulmonary pressure may not be greatly raised to levels approaching the aortic pressure. In the case reported, it was not more than 60% of aortic pressure; in a case (Case VII, Table II) reported by Wood (1950) with a very large shunt, the mean pulmonary artery pressure was 40 millimetres of mercury, well below systemic levels. Presumably with a large communication the usual conditions for turbulent flow which produce the murmur are modified. The same phenomenon is found in cases of large ventricular septal defect, which may have merely a blowing systolic murmur, whereas a small defect may cause the loud murmur and thrill of the *maladie de Roger*. Support for this contention is gained from the fact that in the reported case, when recanalization occurred with the formation of a relatively small channel (as deduced by the normal peripheral pulse and blood pressure and confirmed at operation), a typical continuous murmur became audible.

Fourthly, in cardiac failure a typical continuous murmur heard previously may disappear, presumably because the pulmonary artery pressure rises behind the failing left ventricle, thus altering the usual pressure gradient between the great vessels. The situation is comparable to that in infants. An example is reported by Campbell and Hudson (1952).

Lastly, in the pulmonary hypertension which occurs as a sustained condition independent of age, of size of ductus, or of cardiac failure, and owes its presence to a greatly increased pulmonary vascular resistance, a similar mechanism probably operates. Only a pulmonary systolic murmur may be heard, or there may be no murmur at all; the early diastolic murmur of pulmonary incompetence is often present. This situation is illustrated by the second and third cases reported.

Under these circumstances, how can we arrive at a clinical diagnosis of patency of the *ductus arteriosus*? The first case reported had been regarded as a case of Lutembacher's syndrome, the combination of atrial septal defect and mitral stenosis. This diagnosis was made because of the marked mid-diastolic murmur and thrill at the apex, and the evidence of left auricular enlargement, although the waterhammer pulse and left ventricular hypertrophy were inconsistent with the syndrome. It should be emphasized that mid-diastolic murmurs are common in congenital left-right shunts of considerable size. In atrial septal defect they are heard frequently, and are by no means always associated with mitral valve disease (Nadas and Alimurung, 1952). They occur, too, in both patent *ductus arteriosus* and ventricular septal defect, especially where the shunt is considerable. Here they are ascribed to the effect of the greatly increased flow of blood through the mitral valve (Wood, 1950). Thus the finding of a mid-diastolic murmur or of left auricular enlargement should not automatically lead to the diagnosis of Lutembacher's syndrome, but rather to the suspicion that a large left-right shunt is present which requires careful investigation.

The most difficult clinical differential diagnosis is from a ventricular septal defect, especially if, as sometimes happens, the latter lesion is associated with aortic incompetence. The classical *maladie de Roger*, with a loud harsh systolic murmur and thrill heard best in the fourth left intercostal space near the left sternal edge, presents little or no difficulty. But in other cases of ventricular septal defect, especially when the shunt, and presumably the defect, are of large size, this murmur and thrill are absent. There may be a systolic murmur of blowing quality along the left sternal edge and in the pulmonary area, which may be accompanied at the pulmonary area by the early diastolic murmur of pulmonary incompetence. In this type of case there is usually a mid-diastolic murmur at the apex. There is clinical and electrocardiographic evidence of left as well as right ventricular enlargement. The pulse is usually small in quality, but if the aortic cusp of the aortic valve is involved in the anomaly, there may be aortic incompetence with a waterhammer pulse and wide pulse pressure (Soulié *et alii*, 1949). Thus the physical signs may closely resemble those of the first two cases presented here; certain differentiation is possible only by cardiac catheterization.

The problem is important not only in regard to older children, but in regard to infants, in whom both large ventricular septal defects and on occasions a large patent *ductus arteriosus* may lead to great cardiac enlargement and to cardiac failure (Powell, 1953). Although a continuous murmur may be heard in cases of patent *ductus arteriosus* in infancy before the age of one year (Ziegler, 1952), in others only a systolic murmur may be present. These include not only the common relatively small ductus, but a proportion of the cases in which there is a large shunt, and these it is important to recognize. Dammann and Sell (1952) have reported 24 suspected cases from the Johns Hopkins Hospital, the majority of patients being under the age of five years. All had evidence of a large left-right shunt, with cardiac enlargement and pulmonary plethora, and all underwent thoracotomy, some after preliminary catheterization. Fifteen of the patients were found to have a large patent *ductus arteriosus*, two an aorto-pulmonary septal defect, and seven a ventricular septal defect. Yet the physical signs in the groups were apparently indistinguishable. There was no one reliable differentiating feature. The peripheral pulse, for instance, was recorded as "bounding" with a high pulse pressure in 21 of the 24 cases, including several cases of ventricular septal defect; yet it was normal in at least one of the cases of patent *ductus arteriosus*. The authors concluded that the sign was "of no diagnostic aid". This may be overstating the case somewhat, but it does emphasize the difficulty of establishing a certain clinical diagnosis. In view of the occurrence of recanalization in Case I, it is interesting to note that in Dammann and Sell's cases of patent *ductus arteriosus* a continuous murmur appeared some months after operation in three instances, owing also to presumed recanalization.

The diagnosis of primary pulmonary hypertension may be entertained in cases in which there are signs of considerable pulmonary hypertension; but in subjects who remain clinically acyanotic and have some persistent left-right shunt there is usually enough evidence of left ventricular enlargement to make the diagnosis improbable. The patient in Case II, for instance, had a normal left ventricular type of cardiac impulse, and an electrocardiogram which showed only slight right ventricular dominance. The finding of pulmonary plethora on X-ray examination suggested the presence of a left-right shunt in addition to pulmonary hypertension.

It should be emphasized that in any case of congenital heart disease without cyanosis, in which a left-to-right shunt is suspected, unless the findings are clearly those of the *maladie de Roger*, any suggestion of left in addition to right ventricular enlargement, and any suggestion of waterhammer pulse and wide pulse pressure should lead to careful assessment of the case in order to disprove or to establish the diagnosis of patent *ductus arteriosus*. Apart from operation, this can be done with any certainty only by cardiac catheterization. This investigation can prove the presence of a left-right shunt, and show its site to be aorto-pulmonary rather than between ventricles or auricles, although some care must be taken in the interpretation of results in the presence of pulmonary incompetence. It may be possible further to pass the catheter through the ductus, and thus prove the site of the shunt to be via a patent ductus rather than the much less common aorto-pulmonary septal defect. The differentiation of the latter lesion has been made by retrograde aortography, but it is probable that thoracotomy is a more satisfactory diagnostic procedure in this case, with little more hazard.

It should be pointed out that the patients under discussion have definite evidence of heart disease apart from the mere presence of a pulmonary systolic murmur; there is evidence of cardiac enlargement, and there are abnormal electrocardiographic and radiological findings. Although it is possible that a patient with a pulmonary systolic murmur as the only abnormal physical sign may be found to have a small patent ductus—Gross and Longino (1951) state they have seen it occasionally—the diagnosis can scarcely be of much practical importance. The cases in which diagnosis is essential—those in which a large patent ductus or severe pulmonary hypertension is present—will provide more definite indications of a heart lesion, although its true nature may be obscure.

The third case represents another variety of presentation of patent *ductus arteriosus*. Here there has been sufficient associated pulmonary hypertension to cause definite reversal of shunt and clinical cyanosis, occurring early in life and not apparently progressive. The clinical features can be included in the so-called Eisenmenger syndrome, in which pulmonary hypertension is associated with reversal of flow through ventricular or atrial septal defects, or a patent *ductus arteriosus*. As it is thought inadvisable to ligate this type of patent *ductus arteriosus* (Dammann *et alii*, 1953) the diagnosis of the exact site of shunt does not have great practical importance. The general differentiation from Fallot's tetralogy and pulmonary stenosis with patent *foramen ovale* has to be made. That the shunt is via a patent *ductus arteriosus* may be indicated by more pronounced cyanosis and clubbing of the toes compared with the fingers, and lower oxygen content of samples from the femoral artery than from the right brachial samples. Angiocardiography may show early opacification of the descending aorta. In the case reported, the cardiac catheter demonstrated the site of the defect directly as it passed from the pulmonary artery into the aorta via the patent *ductus arteriosus*.

One point of considerable interest is the fact that all these patients had a history of maternal rubella in the early months of pregnancy. Although the incidence of such a history is not common in cases of patent *ductus arteriosus* taken as a whole, yet when it is obtained the likelihood is strong that the lesion is or includes a patent *ductus arteriosus*. Jackson (1950), examining the histories of 347 Melbourne children born between 1942 and 1949 in whom a congenital cardiac lesion had been proved at operation or autopsy, found seven to have a history of maternal rubella. Of these seven patients, all had a patent *ductus arteriosus*; in six the lesion was uncomplicated.

Essentially similar findings have been reported by Wesselhoeft (1947) and by Gibson and Lewis (1952). It is interesting that in other reported series of atypical patent *ductus arteriosus* the history is found frequently. Thus, of two cases reported by Myers *et alii* (1951), in one there was a maternal history of definite rubella, and in the other a history of possible rubella. A third patient with a rubella history and essentially similar physical findings was found at operation to have an aorto-pulmonary septal defect. In a recent series of eight cases of patent *ductus arteriosus* with pulmonary hypertension reported by Hultgren *et alii* (1953), of the four acyanotic patients one had a definite rubella history, and another had bilateral congenital cataracts, although no history of maternal rubella is mentioned. Hence a history of maternal rubella in the early months of pregnancy is a good indication to suspect a patent *ductus arteriosus*, even if, as in the three cases reported, no classical continuous murmur is present.

Summary.

Three cases of patent *ductus arteriosus* are reported in which the clinical signs were atypical; in particular, the classical continuous murmur was absent. In all the diagnosis was made in late childhood; in all there was a history of maternal rubella. The first patient had a very large ductus; the others had severe pulmonary hypertension, one without and one with cyanosis, the former being treated surgically with success.

The circumstances under which a continuous murmur may not be heard in patent *ductus arteriosus* are enumerated, and the difficulties of differential diagnosis are discussed. It is considered that in any case of suspected congenital left-right shunt, apart perhaps from the classical *maladie de Roger*, if there is found evidence of left ventricular enlargement, or of wide pulse-pressure and a waterhammer pulse, or of a maternal history of rubella, careful assessment and investigation are warranted to prove or disprove the presence of a patent *ductus arteriosus*. Cardiac catheterization will probably be necessary to arrive at a certain diagnosis.

Acknowledgements.

My thanks are due to Dr. C. J. Officer Brown for permission to publish the histories of these patients under his care, to Dr. J. R. E. Fraser for help in their investigation, and to Miss J. Upfill, B.Sc., for technical assistance.

References.

CAMPBELL, M., and HUDSON, R. (1952), "The Disappearance of the Continuous Murmur of Patent Ductus Arteriosus", *Guy's Hosp. Rep.*, 101 : 32.

DAMMANN, J. F., BERTHRONG, M., and BING, R. J. (1953), "Reverse Ductus", *Bull. Johns Hopkins Hosp.*, 92 : 128.

DAMMANN, J. F., and SELL, C. G. R. (1952), "Patent Ductus Arteriosus in the Absence of a Continuous Murmur", *Circulation*, 6 : 110.

GIBSON, G. A. (1900), "Clinical Lectures on Circulatory Affections—I. Persistence of the Arterial Duct and Its Diagnosis", *Edinburgh M. J.*, 8 : 1.

GIBSON, S., and LEWIS, K. C. (1952), "Congenital Heart Disease Following Maternal Rubella During Pregnancy", *Am. J. Dis. Child.*, 83 : 317.

GROSS, R. E., and LONGINO, L. A. (1951), "The Patent Ductus Arteriosus", *Circulation*, 3 : 125.

HULTGREN, H., SELZER, A., PURDY, A., HOLMAN, E., and GERBODE, F. (1953), "The Syndrome of Patent Ductus Arteriosus with Pulmonary Hypertension", *Circulation*, 8 : 15.

JACKSON, A. V. (1950), "Congenital Heart Disease and Maternal Rubella", in "Studies in Pathology", Melbourne University Press : 255.

MYERS, G. S., SCANNELL, J. G., WYMAN, S. M., DIMOND, E. G., and HURST, J. W. (1951), "Atypical Patent Ductus Arteriosus with Absence of the Usual Aortic-Pulmonary Pressure Gradient and of the Characteristic Murmur", *Am. Heart J.*, 41 : 819.

NADAS, A. S., and ALIMURUNG, M. M. (1952), "Atrial Diastolic Murmurs in Congenital Heart Disease", *Am. Heart J.*, 43 : 691.

POWELL, M. L. (1953), personal communication.

SOULIÉ, P., ROUTIER, D., and BERNAL, P. (1949), *"Communication interventriculaire avec insuffisance aortique"*, *Arch. mal. cœur*, 42 : 765.

TAUSSIG, H. B. (1947), "Congenital Malformations of the Heart", New York, The Commonwealth Fund : 331.

WESSELHOEFT, C. (1947), "Rubella (German Measles)", *New England J. Med.*, 236 : 978.

WOOD, P. (1950), "Congenital Heart Disease", *Brit. M. J.*, 2 : 639.

ZIEGLER, R. F. (1952), "The Importance of Patent Ductus Arteriosus in Infants", *Am. Heart J.*, 43 : 553.

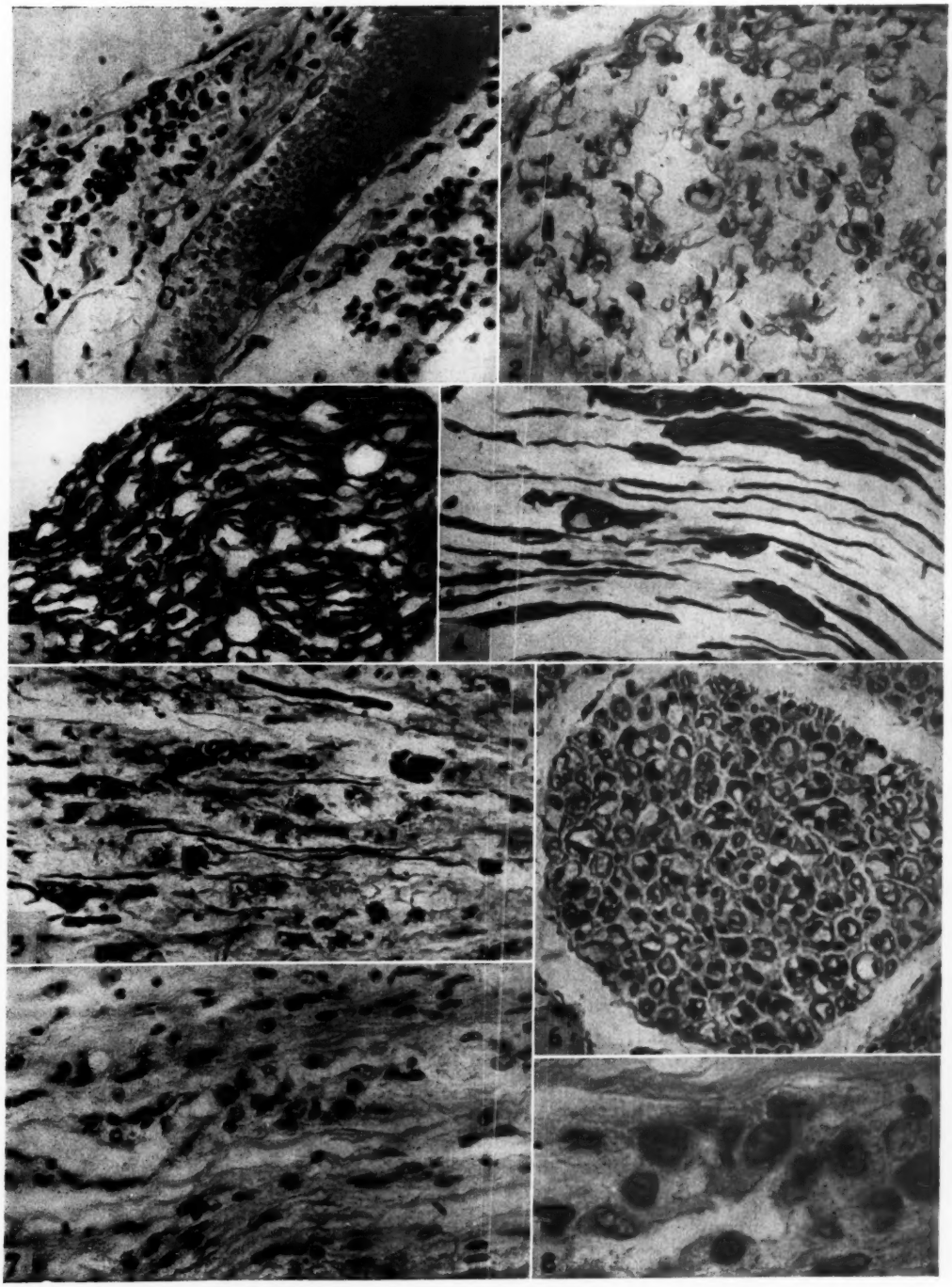
Reviews.

Practical Procedures in Clinical Medicine: Biochemical and Radiological Investigations. By R. I. S. Bayliss, M.A., M.D. (Cantab.), M.R.C.P.; Second Edition; 1954. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 500, with 61 illustrations. Price: 32s.

THIS book was first published in 1950 and a second edition after only four years speaks well for the way the book has been received, especially as the subject matter is not of the type that rapidly becomes out of date. A useful addition to this edition is a short list of references of the right type at the end of each chapter.

As previously, the chapters deal separately with the various systems, and there are special chapters on blood transfusion, parenteral fluid therapy, radiology, and miscellania. The order of each section is good: (*a*) Physiological and Biochemical Principles, (*b*) Investigations Used and Indications, (*c*) Requirements, (*d*) Details of Technique, (*e*) Interpretation. An adequate consideration of these factors in each case at the bedside should reduce unnecessary investigations and make those carried out more useful.

ILLUSTRATIONS TO THE ARTICLE BY R. D. K. REYE.



ILLUSTRATIONS TO THE ARTICLE BY J. M. GARDINER.

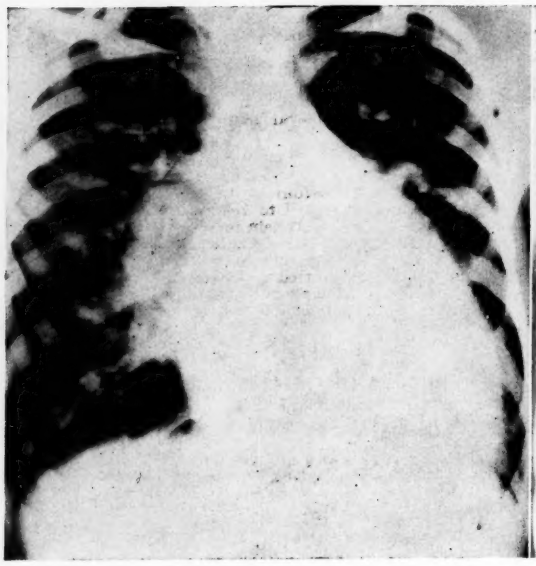


FIGURE IV.

Postero-anterior X-ray film of the chest in Case I, showing large heart and pronounced pulmonary plethora.

FIGURE V.

Postero-anterior X-ray film of the chest in Case II before operation, showing prominent main pulmonary artery and slight pulmonary plethora.

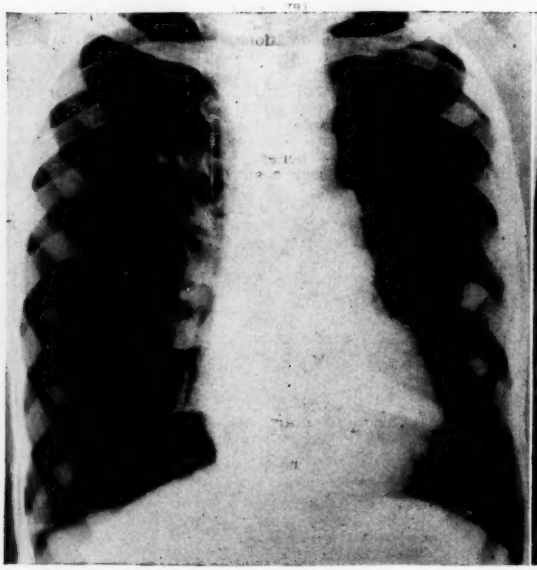


FIGURE VI.

Postero-anterior X-ray film of the chest in Case II after operation, showing diminution in heart size, especially of the main pulmonary artery.

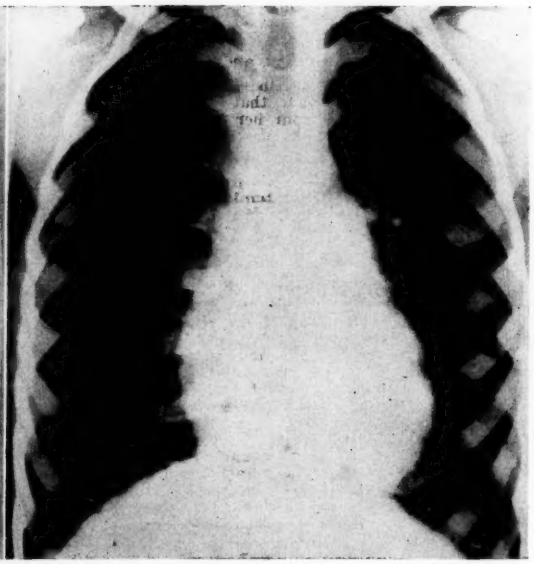


FIGURE VII.

Postero-anterior X-ray film of the chest in Case III, showing slight cardiac enlargement with prominence of the main pulmonary artery.

The chapter on miscellania is particularly useful and contains a fund of information on all kinds of topics, but information often required at short notice.

We consider that there is one serious omission in this book, which otherwise is so complete. There is insufficient reference to modifications of procedures when used in the case of infants and children. Many procedures used only in pædiatrics are not described at all. For example, the following topics could well be discussed: (i) parenteral fluid therapy in relation to babies, (ii) lumbar puncture in babies, (iii) collection of urine in babies, (iv) details of diets for babies. Pædiatrics is very much part of clinical medicine, and these types of procedure are often those not generally familiar to resident medical officers and practitioners.

This book is filling a gap in medical literature and should be available to students, "residents" and practitioners at the bedside, thus assisting with many details of technique and interpretation, which are often a burden to attempt to remember. We shall look forward to further editions of this book, anticipating an even better production.

A Textbook for Midwives. By Margaret F. Myles, S.R.N., S.C.M., H.V.Cert., Sister Tutor Cert., M.T.D., with a foreword by Jean P. Ferlie, O.B.E., R.G.N., S.C.M.; 1953. Edinburgh and London: E. and S. Livingstone, Limited. 8¼" × 6", pp. 688, with 380 illustrations. Price: 42s.

THIS book should be extremely well received by midwifery tutors and pupils alike as an excellent publication abundantly illustrated. The author has had experience not only in the United States of America, but principally in the Simpson Memorial Maternity Pavilion in Edinburgh, from where she wrote the book.

It is easy reading and contains more than the minimum requirements for the Central Midwives Board examinations, so that it may serve as a complete reference manual for midwives. Each chapter is well set out and stresses basic principles, and at the end of each is a set of test questions.

The ante-natal section is full, and proper stress is placed on the preparation of the mother, whilst the father is not neglected. The administration of "Avertin" as a basal sedative in eclampsia is given prominence, as this is a popular form of therapy in Scotland. The modern classification of *placenta prævia* is given preference over the older, whilst the description of the third stage of labour is a delight to read. Labour is classified as prolonged when it has exceeded twenty-four hours. There is a section on mothercraft teaching, giving much detail and illustration, and tutors should gain much help from the author's practical teaching hints.

The last section in the book is intriguing, as it includes a short history of midwifery from prehistoric times to the present day, as well as appendices on domiciliary midwifery, sterility, carcinoma in pregnancy, and vital statistics.

Mrs. Myles is to be congratulated on her presentation, which cannot conceal the fact that she is a teacher well able to transmit teaching from her wide experience. The book could well be read by graduates of medicine.

Ten Lepers from Naestved in Denmark: A Study of Skeletons from a Medieval Danish Leper Hospital. By Vilhelm Møller-Christensen, M.D., with a preface by Erik Waaler; 1953. Copenhagen: Danish Science Press, Limited. 10¼" × 7", pp. 162, with 119 illustrations. Price: $5.00.

BETWEEN the years 1941 and 1953 the author had been engaged in a systematic excavation and examination of the burial grounds of the Æbelpolt Monastery in North Sealand, which flourished between 1175 and 1544. In 1944 the skeleton of a woman, aged twenty-five to thirty years, presented changes that suggested they might be due to leprosy. In the Middle Ages Denmark had at least 35 leper hospitals, called Saint Jorgen's Hospitals. The site of one of these at Næstved in Southern Sealand showed that about 200 persons had been buried there. This work contains a detailed description of the pathological changes in the skeletons of ten of these lepers. As a result of his examination, the author describes "a hitherto entirely unknown complex of pathological changes localized to the bones of the face and to the legs, apparently specific of leprosy". The changes in the skull consist of atrophy of the anterior nasal spine, giving rise to an appearance that he has designated as *"facies leprosa"*. By means of radiography this atrophy was shown to be present in five out of seven living lepers in Bergen, and as it appears to be a useful new sign in the diagnosis of leprosy, he has called it the Bergen symptom. In the bones of the legs, enlargement of the nutrient canals in four cases was believed to be due to a leprous endarteritis and the presence of cysts in the bones to the direct action of Hansen's bacillus. It would be interesting to ascertain whether any of these changes can be detected in Australian cases of leprosy.

Dr. Møller-Christensen's book is well written in English, and contains 119 illustrations, mostly of the changes in the bones, but also of the farmyard that occupied the site of the Saint George's Hospital, the appearance of skeletons *in situ*, a map of the situation and a tentative ground plan of the hospital. On page 131 Australian natives are unfortunately called "Australian Negroes". The work is dedicated to the three great pioneers in the fight against leprosy in Norway—Boeck, Hansen and Danielsen—portraits of whom appear after the title page. Those in charge of lepers in Australia will find much of interest in this book, and we recommend its study to them. It is also of interest to the pathologist, the mediæval historian, and even the general practitioner.

It is interesting to note that Saint Jorgen's farm, whose inhabitants and stock obtained water from a well on the premises, supplied customers with milk—not that there could be any danger after 400 to 700 years. The skeletons, with one exception, lay with their feet towards the east and their heads towards the west. According to an old Scandinavian custom, criminals were buried in the reverse position. The one skeleton here buried in reverse was that of a woman aged thirty. It is pathetic to think that in these hard old times social condemnation even followed the corpse to the grave.

The Psycho-Analysis of the Nursery. By Alice Balint; 1953. London: Routledge and Kegan Paul, Limited. Sydney: Walter Standish and Sons. 8¼" × 5¼", pp. 156. Price: 12s. 6d.

TWENTY-THREE years ago the late Alice Balint published a book in Hungarian. Now it appears in English under the title of "The Psycho-Analysis of the Nursery". It is a simple and unpretentious account of the young child's behaviour in the nursery and through the early formative years. Though fully flavoured with Freudian ideas, it is neither dogmatic nor too technical. Unlike some translations, it is easy to read. It is informative and obviously sincere.

Mrs. Balint, whose academic interests included pedagogy, anthropology and psychoanalysis—including the bringing up of children—found the nursery the place where pre-history and civilization meet, when the primitive is transformed into the civilized being. Her story centres round this transformation, so often fraught with tears and frustrations, and so often the locus of later neuroses. She illustrates the child's Œdipus difficulties, his castration anxieties, his repressed fantasies and identifications, with personal examples in a way which not only smooths out the difficulties, but overcomes the indelicacies of a subject which to many is still somewhat unpalatable. Her calm and persuasive manner will allow the lay reader to feel that he is treading on solid ground. There is nothing new here for the psychoanalyst or the child psychiatrist; but the teacher, the nurse and the parent might surely benefit from the perusal of a book which deals plainly with a subject of perennial interest and which is not too long.

Diseases of Women. By Robert James Crossen, A.B., M.D., F.A.C.S.; Tenth Edition; 1953. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 10¼" × 7", pp. 948, with 990 illustrations, 41 in colour. Price: £9 14s. 3d.

THE tenth edition of this book takes on a new aspect, since it is revised by R. J. Crossen only, after the death of his famous father, Harry Sturgeon Crossen. The standards and quality of the work do not suffer, and at least for the graduate they are improved. The student perhaps may be confused by the elementary and the advanced post-graduate teaching often intermingled on the same page.

Two "guest writers" have contributed chapters. It is to be hoped this practice does not grow.

A striking feature of great value to the graduate is the copious list of references. The author quotes extensively from sources both modern and not so modern, but always to the point.

The paper and printing are excellent; the style is easy. Illustrations are profuse and mainly good, and Crossen does not hesitate to use old and well-tried line drawings, in which the salient points are easily grasped.

In our review of the eighth edition we strongly criticized the authors' preference for subtotal hysterectomy over total hysterectomy. This criticism is now not necessary. The

total hysterectomy seems to have supplanted the subtotal. The author also advises the removal of ovaries as well in women approaching or beyond the menopause. Some operations are described in detail, but usually the reader is referred to the companion volume on operative surgery.

Excellent chapters include those on gynæcological examination and diagnosis, psychosomatic gynæcology, menstrual disorders, and uterine cancer. Cervical fibroid tumours are dismissed in five lines, torsion of ovarian tumours in little more, whilst brucellosis and actinomycosis are discussed at length.

A genuine attempt seems to have been made to revise some of the obsolete teaching handed down from text-book to text-book. For example, we thoroughly agree that the reposition of a retroverted uterus by an intrauterine sound is dangerous and should be condemned.

We quote the following extract without comment: ". . . in many countries in which this text is used the antibiotics are not generally available."

In discussing the diagnosis of ectopic gestation Crossen writes: "Curettage may be helpful." It certainly would help the diagnosis, but not the embryo, if instead of a suspected ectopic a threatened miscarriage was present. Also we do not agree that in all cases of extensive endometriosis drainage should be carried out. Watchful expectancy is advocated in mild or non-increasing intraperitoneal collections of blood. We would not quarrel with the gynæcologist who explores the abdomen in any definite intraperitoneal hæmorrhage.

Most of our differences with the author are minor ones. This is a very good gynæcological text-book.

The Chest: A Handbook of Roentgen Diagnosis. By Leo G. Rigler, M.D.; Second Edition; 1954. Chicago: The Year Book Publishers, Incorporated. 8" x 5¼", pp. 380, with 338 illustrations. Price: $8.00.

THE new edition of this book was considered necessary because of advances in Röntgen diagnosis work, and many new illustrations have been included and others replaced. The author points out that in practically all diseases there are some manifestations in the chest. Great stress is laid on fluoroscopy, and quite an elaborate programme is set out. In a busy practice this would mean considerable exposure to dangerous radiation to the operator. Tomograph examination has been more widely used in chest work of late. The technique of radiography is described in detail. The whole work is worth study, but the section on the normal chest is particularly worthy of attention. The rest of the work is of the "atlas" type—a maximum of illustration with a minimum of text. However, the descriptive matter is quite ample. The illustrations are of the highest quality. The author insists on a full history being obtained in all cases, and particularly in cases of industrial diseases. Some interesting films of pneumonitis and fibrosis following irradiation are included. It is pointed out that in sarcoidosis the extensive lung changes are out of all proportion to the symptoms. Improved methods of bronchography are described. This is a very useful little book of handy size and is well worth careful study.

Anoxia of the New-Born Infant. A symposium organized by the Council for International Organizations of Medical Sciences established under the joint auspices of U.N.E.S.C.O. and W.H.O.; Consulting Editors, Dr. Kenneth W. Cross (U.K.), Dr. Marcel Lelong (France) and Dr. Clement A. Smith (U.S.A.); Editors for the Council, J. F. Delafresnaye (France) and T. E. Oppé (England). Oxford: Blackwell Scientific Publications. 8½" x 5½", pp. 248, with many illustrations. Price: 27s. 6d.

THIS monograph is a collection of the papers read at the symposium held in London in October, 1951. There were 17 participants, all of international reputation, gathered from the United Kingdom, United States of America, and several European countries. These included pædiatricians, obstetricians, pathologists, physiologists, anatomists and chemists. It is stated at the outset that no attempt has been made to cover the complete field of neo-natal anoxia, but that rather each worker has presented some aspect to which personal contribution had been made. There is a report also of the discussions which followed the reading of the papers. This is a highly stimulating and informative publication, particularly for pædiatricians and obstetricians. It includes a paper on the recognition of fœtal distress, which, according to studies of the heart sounds made by M. Mayer with an electronic pick-up microphone, is evidenced not by tachycardia, but by slowing and arrhythmia. This change is said to be particularly important if present in the first stage of labour. G. F. Gibberd, discussing the prevention of anoxia, lays great stress on the simple and often neglected precaution of maintaining the head-down position of the fœtus, and records that 50 to 80 millilitres of fluid may run from the mouth in this position.

M. Lelong and R. Laumonier bring forward a theory of development of the lungs, based on histological examination of 62 abnormal lungs, which is not convincing. A. Rossier's description of the prolonged and intensive oxygen therapy considered necessary for Parisian premature infants is surprising.

The "hyaline membrane" is discussed by Clement Smith, Agnes Macgregor, M. Lelong and R. Laumonier, and E. H. Ahvenainin. No final agreement on the ætiology was reached. R. Brinkman and J. H. Jonxis make contributions to the physiological side of anoxia, and K. Cross and T. Oppé discuss the implication of the neonate's response to high and low oxygen atmospheres. J. Walker's figures on the oxygen levels of human cord-blood during pregnancy and during normal and abnormal birth are of special interest, and it is gratifying to note his emphasis on the potential danger to the fœtus of post-maturity. This should convince the sceptics. J. Lind produces some excellent plates of angio-radiography in newborn intact fœtuses of 18 to 22 weeks, confirming for the human the work of Barclay *et alii* with lambs. He states that open *via sinistra* often occurs in asphyxia. Finally, there are published the eight and a half pages of points on which the speakers were agreed, amongst which it is refreshing to find the emphasis laid on simple procedures for resuscitation.

Bailey's Text-Book of Histology. Revised by Philip E. Smith, Ph.D., Sc.D., and Wilfred M. Copenhaver, Ph.D., with the assistance of Dorothy D. Johnson, Ph.D.; Thirteenth Edition; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" x 6½", pp. 794, with 442 illustrations, a few in colour. Price: £4 16s. 9d.

FIRST published in 1904, "Bailey's Histology" has now reached its thirteenth edition. It would be interesting to have a copy of that small book of 1904 to compare with the present large and beautifully illustrated volume. Text-books of histology are becoming more and more elaborate. As the revisers point out, even in the short time which has elapsed since the twelfth edition was published in 1948, there have been marked advances in the field of histology, particularly in histochemistry, in electron microscopy and in the elucidation of structural-functional relationships. As this book is intended primarily for the use of first-year students in medicine and dentistry, the revisers have not attempted to include all of this "new research material" but have selected what seems suitable for their purpose. To do this, changes in the text have been made throughout most of the book and some sections have been entirely rewritten. Six new colour plates, most of which contain several individual illustrations, have been added, and also 88 half-tone and line figures, some of which replace those in previous editions. The illustrations include a few electron micrographs. Most of the photomicrographs and other illustrations are very good indeed, but, curiously enough, Figures 74 and 76, showing the cells of the blood, are rather poor colour drawings.

The keynote of this book is the indissoluble relationship between structure and function; the authors maintain their principle that a "knowledge of the structure and activities of the cell must necessarily form the basis of histology". Their book is well written and most readable; it makes histology live. The paper, type and production are excellent. One realizes that these good qualities must be paid for, but a fact that must give most students pause is that while the last edition cost £1 12s. 6d. here, the price of the present one in this country is £4 16s. 9d. The ever-increasing cost of text-books is a serious problem.

Handbook of Tropical Dermatology and Medical Mycology. Edited by R. D. G. P. H. Simons; 1953. Amsterdam: Elsevier Publishing Company. London: Cleaver-Hume Press, Limited. Volume II. 9" x 6½", pp. 862, with 510 illustrations. Price: £5 each volume.

THIS volume of the "Handbook of Tropical Dermatology and Medical Mycology" maintains the commendable standard of the first volume of the work and satisfactorily completes the ambitious aim of dealing with every tropical skin affection of importance. In this volume the editor is assisted by a group of 56 contributors from various countries. His difficult editorial task, complicated by the necessity for much translation, is well performed, and uniformity and balance obtain throughout the large work. The text is amplified by numerous illustrations.

The volume opens with a section on diseases due to animal parasites. In this a chapter on *filariasis bancrofti*, by Sir Philip Manson-Bahr, is of special interest to workers in this region. The next section, devoted to fungous diseases, occupies over 400 pages and provides a comprehensive treatment of the subject. In addition to clinical discussion of the many types of fungous infections, chapters deal with the physiology of fungi, mycological technique, experimental infections, therapy and cultural characteristics. There follows a section on diseases due to allergy, malnutrition, and metabolic disturbances. This includes a particularly interesting chapter on clinical and biochemical features of the skin in malnutrition, by Professor B. S. Platt, of London. The greater part of an important section on tumours of the skin, dealing with malignant conditions, is contributed by two Sydney workers, Dr. John C. Belisario and Dr. R. E. J. ten Seldam. A chapter on ætiology is the joint work of these authors, while that on symptomatology and treatment is by Dr. Belisario, and pathology by Dr. ten Seldam. The volume is completed by a section on odds and ends, which includes a chapter on the ship's surgeon and tropical skin affections.

This is an important work, which is highly recommended to tropical physicians and students of tropical medicine, as well as to specialists in temperate climates. It is an essential addition to all medical libraries.

Surgery for Dental Students. By Michael F. A. Woodruff, M.D., M.S. (Melb.), F.R.C.S. (Eng.), with a foreword by S. H. Wass, M.S. (Lond.), F.R.C.S. (Eng.); 1954. Oxford: Blackwell Scientific Publications. 9" × 6", pp. 334, with 108 illustrations. Price: 30s.

DURING the early years of this century dentistry evolved from a craft to a science. Such an evolution demanded a new conception in the training of dental students; furthermore, as the integration of dentistry into the wide field of medical science took place, the necessity for expanding the training of dental students in fundamental subjects became obvious. For example, it was generally agreed that a sound knowledge of the principles of medicine and surgery was necessary for the future entrants to the dental profession. Only in recent years have a few among the many British medical authors attempted to cater for the tremendous change in the training of dental students by providing text-books specifically designed for the dental course. So it is with pleasure that one is introduced to Professor Woodruff's book "Surgery for Dental Students". Being written by a surgeon who is a trained and experienced academic teacher, it incorporates many aspects important to clinical instruction which are frequently left out of standard text-books on surgery.

A number of minor points invite comment. In a text-book designed for dental students meticulous attention should be given to dental and oral anatomy, and in this regard there are a few bad lapses. For example, Figure 39 depicts teeth which in form and arrangement would be more correctly portrayed in the mouth of some animal rather than a human being; also the anatomical proportions of the mandible illustrated in Figure 23 are, to say the least, unusual.

The writer quite rightly condemns the use of the "four-tailed bandage" for first-aid treatment of a fractured mandible, but there is no illustration of the barrel bandage, which is recommended and which is the one most commonly used for the support of the mandible in present-day practice. The section on hare-lip and cleft palate is simply and clearly set out and represents the knowledge on this subject essentially required by the dental student.

The quality of the illustrations is uniformly good, and one is impressed with the over-all excellence of Professor Woodruff's achievement. However, while the principles of general surgery are well set out and illustrated, there is something lacking—namely, a working knowledge of the mouth from the point of view of clinical dental practice. This may or may not be a fair point of criticism, but its rectification would certainly improve the volume in the eyes of those readers for whom it has been especially written.

Methods in Medical Research. Editor-in-Chief, J. Murray Steele; 1954. Chicago: The Year Book Publishers, Incorporated. Volume VI. 8½" × 5½", pp. 284, with 49 text figures. Price: $7.00.

"THERE are men that will make you books and turn them loose into the world with as much dispatch as they would do a dish of fritters" (M. De Cervantes, *Don Quixote*). Thus quotes J. Murray Steele, editor-in-chief of Volume VI of "Methods in Medical Research", who hastens to reassure his readers that this book is the antithesis of such a one. We wholeheartedly agree with Dr. Steele. This volume has been well prepared and is a worthy successor to its five predecessors. It includes sections on "Some Methods of Studying Human Genetics" (editor, A. Ciocco), "Methods in Environment Medical Research" (editor, R. G. Daggs), "Statistics in Medical Research" (editor, D. Mainland), and "Design and Construction of Metabolism Cages" (editor, A. Lazarow). Perhaps the most fascinating section to the practising physician who takes a keen interest in medical research is the one containing the four chapters written by Dr. C. C. Li, University of Pittsburgh, Pennsylvania, on the methods of studying human genetics. Dr. Li patiently and lucidly introduces his readers to the intricacies of human genetics—the genes, their methods of transmission and their effects on both the individual and on the community. He stresses the importance of the mathematical approach to assess the significance of clinical findings. The study of siblings of affected parents in such conditions as *diabetes mellitus* or sickle-cell anæmia has led us to appreciate the genetic factor in disease. The intermarriage of near relatives tends to expose latent traits, both for good and for ill. The reader of this volume will also be interested to learn from Dr. D. Mainland and his colleagues of the ever-increasing influence of statistics in medical research, and of the problems which face the research worker of today. The medical profession as a whole has always been most sympathetic and helpful to those of their colleagues who have chosen to devote their lives to clinical and basic medical research, and this volume will go far to enrich this healthy productive attitude of mind. Moreover, this volume may encourage more general practitioners and consultants to play their part in the practice of systematic clinical investigation. Let us not forget the classical observations of James Mackenzie in Scotland or of Pickles in England.

Chemotherapy of Infections. By H. O. J. Collier, B.A., Ph.D., M.I.Biol., with a foreword by Alexander Fleming; 1954. London: Chapman and Hall, Limited. 7½" × 6", pp. 264, with 53 illustrations. Price: 18s.

THIS pleasant little book, which comes from the series "Frontiers of Science" and is published by Chapman and Hall, contains a surprising amount of solid fact on the subject of antibiotics within its 215 pages. The author begins with an explanatory chapter outlining the levels at which he proposes to discuss his subject, and defines terms and chemical formulæ and measurements which he proposes to use. However, it is not quite clear for whom he intends the book, as in one place he defines the milligramme and the cubic centimetre and then goes on to discuss the host-parasite relationship in disease without defining terms such as toxin or therapeutic substance, although it is true that meanings rapidly become apparent. There is discussion of the host-parasite relationship and disease in man and of the body's defences, the additional weapon of a drug harmless to man but poisonous to the parasite, and the ways in which the parasite reacts to the poison—the development of the phenomenon of drug resistance.

The commoner antibiotics are described in detail and such portions of the mechanism of their action as are understood are mentioned briefly. The chemotherapy of tuberculosis, leprosy, malaria, and the spirochætal diseases is treated in greater detail, and the author concludes with an admirable chapter summing up the present position of knowledge, the need for caution, and lines of future development.

The dust jacket announces that this book will be available from any bookseller. In the light of that statement one can prophesy that the general reading public will bring interest but not understanding to its pages. The biologist and the student and practitioner of medicine on the other hand will find it full of information and facts, while teachers in the subject will find it useful as a reference book. The graphs, tables and formulæ are admirably clear, but some of the diagrams in black and white are not very helpful—that of a phagocyte squeezing between two cells of intestinal epithelium in particular could only be mystifying to the general reader; and other illustrations of bacteria and cells without any labelling marks, only general legends below, also are of little use.

The book is of an unusual size, the type face is excellent, and the paper is of good quality. One should like to browse amongst the other titles in the series.

Rorschach Responses in Old Age. By Louise Bates Ames, Ph.D., Janet Learned, Ph.D., Ruth W. Métraux, M.A., and Richard N. Walker, M.A.; 1954. New York: Paul B. Hoeber, Incorporated, Medical Book Department of Harper and Brothers. 9½" × 6½", pp. 244, with three text figures. Price: $6.75.

IN "Rorschach Responses in Old Age" Dr. Louise Bates Ames, who is Director of Research at the Gesell Institute

of Child Development, and her colleagues present a study in the responses of 200 men and women between the ages of 70 and 100 years. Their work on child Rorschach responses was published in 1952. Their subjects were divided into three age groups, the seventy, eighty and ninety year olds, and from the mental standpoint into the preseniles, seniles and normals. All the subjects were "reasonably alert and healthy", individuals with aberrant behaviour and grossly incompetent being excluded. A third of the subjects were living at home, the remainder were in institutions for the aged. The preseniles gave fewer responses than the normals, with more animal than human movements; perception and expression were vague, and suspicion and insecurity were apparent. Both intellectual and emotional responses were restricted and introversive in trend. The seniles gave animal responses and especially sex parts predominantly in proportion to their deterioration. The responses in general indicated lack of motivation and great limitation of adaptability. With emotional lability there was no real, deep emotion. The authors conclude that after the peak of maturity is passed the direction of development begins to reverse itself. With deterioration the Rorschach responses resemble those of younger persons. It appears, therefore, that the authors confirm what clinical experience and Shakespeare have taught us about "second childishness and mere oblivion". Those who use the Rorschach test will need this work for reference. In these days, when the employability of the elderly is becoming a matter of economic importance, any help that the psychologists can give in the process of selection of persons and jobs is to be welcomed.

The Mothercraft Manual: Or the Expectant and Nursing Mother and Baby's First Two Years. By Mabel Liddiard, C.B.E., S.R.N., S.C.M., M.T.S., with an introduction by Dorothy M. Taylor, M.D., D.P.H.; Twelfth Edition; 1954. London: J. and A. Churchill, Limited. 7¼" × 5", pp. 188, with eight plates and 30 text figures. Price: 5s.

THIS book covers a wide range, from the ante-natal period until the end of the second year of life. As the author states, "the entire plan of the book is based upon the principles taught by the late Sir Truby King", and consequently the preparations and routine described are in accordance with that school of mothercraft. The subject matter of this edition has been rewritten to bring it into line with modern practice, and it is a very readable and useful little volume. As the book is written from experience in England, much of the information provided is not applicable to Australian conditions. This is particularly evident from the chapter upon care of the premature baby in the home—a necessity that arises only rarely in this country.

Entomology (Medical and Veterinary): Including Insecticides and Insect and Rat Control. By D. N. Roy, M.D., D.T.M., D.Sc., and A. W. A. Brown, M.B.E., Ph.D.; Second Edition; 1954. Calcutta: Excelsior Press. 10" × 7½", pp. 422, with 205 illustrations. Price: Rs. 30.

THIS work is divided into two parts, the first of 290 pages, dealing with medical and veterinary entomology, being by Dr. D. N. Roy, who is in charge of the Department of Medical Entomology and Biology, Indian Institute for Medical Research, Calcutta, and the second of 116 pages, dealing with insecticides and insect and rat control, by Dr. A. W. A. Brown, of the University of Western Ontario, London, Canada. Though intended primarily for use in India, and consequently dealing thoroughly with India's medical entomology, giving, for instance, keys to the identification of anopheline larvæ and the mosquitoes of that country, Dr. Roy, as he says in his preface, has incorporated "information on similar lines in respect of other countries" but in briefer form. An indication of the scope of Part I will show how useful this work can be to medical entomologists and to medical and veterinary men practising not only in the tropics, but also in temperate climates. Chapter I has two pages on leeches. Chapter II deals with the structure of insects and a full description is given of the cockroach as a type. Some 80 pages deal with the mosquitoes—larvæ, adults, life histories, the diseases conveyed by them, and their control. Then follow shorter accounts of *Phlebotomus*, *Culicoides*, *Simulium*, and the Tabanids, of the house fly and its control, of maggot infestations, of *Stomoxys* and the Glossinae. Plague and the plague fleas receive, as one would expect, full consideration. Beetles and caterpillar dermatitis are dealt with shortly, but the bugs, lice and ticks get adequate attention.

Dr. Brown's Part II has, of course, been written so that all the world can read with benefit his account of insecticides and insect and rat control. His chapters deal with the chemistry of insecticides, their toxicology, their application, the insecticidal control of noxious diptera, and the control of fleas, lice on man and animals, bed-bugs, cockroaches, mites, ticks and rats.

Many references are given at the end of some of the chapters and a few Australian references are included. There is a reference on page 33 to the virus of equine encephalitis as being present in mosquitoes, but the Australian work on Murray Valley encephalitis has been too recent to receive any notice. Was the original cradle of yellow fever in South America? "Diverticulæ" and "labellæ" are blemishes. The usefulness of this book has been greatly increased by the inclusion in this the second edition of the part dealing with insecticides and vermin control, which appeals strongly to the public health official as well as to medical and veterinary men practising in the tropics.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Roentgenographic Technique: A Manual for Physicians, Students and Technicians", by Darmon Artelle Rhinehart, A.M., M.D., F.A.C.R.; Fourth Edition; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 454, with 216 illustrations. Price: 91s. 6d.

The subtitle defines the scope of the book.

"Histopathology of the Skin", by Walter F. Lever, M.D.; Second Edition; 1954. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 536, with 281 illustrations, eight in colour. Price: £6 9s.

Based on the courses of dermatopathology given in recent years to graduate students of dermatology enrolled at Harvard Medical School and Massachusetts General Hospital, and intended primarily for dermatologists.

"Hypertension and Nephritis", by Arthur M. Fishberg, M.D.; Fifth Edition; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 986, with 49 illustrations. Price: £6 14s. 6d.

The last revision of this important book was made fifteen years ago and it has been entirely rewritten.

"The Year Book of Endocrinology (1953-1954 Year Book Series)", edited by Gilbert S. Gordan, M.D., Ph.D.; 1954. Chicago: The Year Book Publishers, Incorporated. 8" × 5½", pp. 390, with 99 illustrations. Price: $6.00.

One of the "Practical Medicine Series of Year Books".

"Recent Advances in Surgery", edited by Harold C. Edwards, C.B.E., M.S., F.R.C.S., with a foreword by Heneage Ogilvie, K.B.E., M.Ch.; Fourth Edition; 1954. London: J. and A. Churchill, Limited. 8" × 5½", pp. 494, with 157 illustrations. Price: 40s.

The first edition was the work of Sir Heneage Ogilvie, who writes the foreword to this edition. The series of "Recent Advances" is widely known.

"Aids to Histology", by Geoffrey H. Bourne, D.Phil., D.Sc.; Sixth Edition; 1954. London: Baillière, Tindall and Cox. 6½" × 4½", pp. 168, with 59 text figures. Price: 6s.

One of the well-known "Students' Aids Series" which was inaugurated in 1876.

"Bacteriology for Students of Dental Surgery", by R. B. Lucas, M.D., M.R.C.P., D.P.H., and Ivor R. H. Kramer, L.D.S., R.C.S.; 1954. London: J. and A. Churchill, Limited. 8½" × 6½", pp. 270, with 57 illustrations. Price: 22s. 6d.

Designed to give the dental student a general conception of the relationship of bacteria to disease in man and some understanding of the meaning of bacterial infection, "concepts necessary both to the practice of dental surgery and to comprehension of the inter-relationship between this and other branches of medicine".

The Medical Journal of Australia

SATURDAY, SEPTEMBER 4, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE PLACE OF BIOLOGY IN EDUCATION.

It is sometimes said of a man that he was educated at such and such a school and we can recall that certain superior schools for girls have been known as finishing schools. The implication that education ends when the pupil leaves school is not largely held today. All thinking people know that education is a continuous process which goes on throughout the life of the individual. Varying views are held on the purpose of education. One well-known university teacher of a bygone age, when faced with the dogmatic statement that the purpose of education was to teach people to think, denied this with great fervour. He had been discussing with a companion a certain group of university students who held views that differed widely from those that he wished to teach them—views on politics, religion and social life generally. Presumably, if they had held the views which he had wished to instil into them, he might have agreed that the purpose of education was to teach these people to think. It is unfortunately true that many of the best of us are swayed more than we realize by our prejudices. Sir Richard Livingstone has recently said that there are three aims of education. The first is to prepare us, either by a general or by a vocational training, to earn our bread; the second is to give us some understanding of the universe and of men; and the third is to help us to become fully developed human beings. James Bryant Conant has written that education should be regarded as a social progress—it is related in each country to the current political scene, the social history of the nation and the educational needs. This is no doubt true, but the educated man must not only understand his own social *milieu* and its significance, but he must know something about the conditions under which neighbouring peoples live. We may indeed ask whether a man can ever be regarded as educated. Those who have read James Bell on civilization will recall that he would apply the term "civilized" to very few human beings; he regards civilization as being the product of security, leisure and liberty—in other words, those who have to work for a living have no time to cultivate the qualities of the mind which will make them civilized beings. To a certain extent this argument may be applied to education, for who can say when we have advanced to such a stage of knowledge, understanding and breadth of sympathy that it will make us truly educated?

If we are to have some understanding of the universe and of men we must know something about science and particularly about biology. It is more than twenty years since we discussed in these pages the place of biology in education. On that occasion, we insisted that education should aim not only at knowledge of certain happenings (in these circumstances it degenerated into mere instruction), but at understanding. If young people were to be able to cope with life, they ought to be able to take an intelligent interest in it—they should, *inter alia*, be able to realize what was going on around them in the world of plants and animals. We stated that biology would teach them something about the development of life and would bring them as far as the gate beyond which lay the possibility of higher development within themselves. At the same time, biology alone was not sufficient; it was, however, a science which was fundamental and therefore indispensable. The subject has been brought to notice again by an address delivered on the occasion of the opening of the new Hatherly biological laboratories at Exeter by Sir Edward Salisbury on April 23, 1953. This address was published in the *Notes and Records of the Royal Society of London*, January, 1954. Sir Edward Salisbury was opening new laboratories in which the subject of biology could be effectively taught. He said that if the maximum good was to accrue from the efforts of those working in the laboratories and from the material improvement of the circumstances provided by them, biology had to take its proper place not only as a special discipline, but as part of that liberal education that constituted an essential element in a cultured mind. It is this indispensability of biology which should be stressed. Sir Edward Salisbury said that in the teaching of biology, and indeed of most subjects in the curricula of schools and universities, there had, in the past, been far too great a tendency to mistake the imparting of mere information for the inculcation of knowledge. Too much attention was devoted to the collection of bricks and far too little attention to the vision of the dwellings into which they were to be constructed. This tended to develop a community of the well informed rather than men and women of wisdom. The bricks were often regarded as important in themselves, and even brickbats, the half-truths, became the missiles of controversy instead of the elements of constructive achievement through which the superstructure of a richly ornamented life and useful citizenship could be built up. He used a happy phrase when he said that many men and

women when they went out into the world of achievement had a mental equipment comparable to a dump rather than to an edifice. There was an inevitable temptation to instil more and more facts into a syllabus, but it was the ideas which illuminated the facts and what could be deduced from them that was so much more significant than the facts themselves. The understanding of the significance of the relations between organisms and the conditions of their natural environment could alone be founded upon an integrated appreciation of the contributions which each and every direction of scientific approach could make to their analysis. The ecological approach was an important instrument of education, since one was far too prone to speak and to think in terms of individual subjects, regardless of the fact that the unity of knowledge was unbroken and that the biologist, the chemist and the physicist, even the mathematician, were but looking into the same room of the unknown through different windows. Sir Edward Salisbury made one statement which may be directly applied to the practice of medicine: he said that the general practitioner of science was just as essential to real advancement as the specialist, provided the one was constantly seized with the significance of the activities of the other.

Sir Edward Salisbury saw two requisites which he deemed essential to the consummation of a liberal education; these were breadth of sympathy and, above all, mental humility. He declared that there was no one road to mental salvation, either by way of the classics or by way of biology. In the past, we have often referred to workers in special fields wearing blinkers. Sir Edward Salisbury put this in another way when he remarked that in the age of specialization it was all too easy to delve deeper and deeper into our own particular furrow, so that almost before we were aware, the banks of our overburden had cut off all view other than a narrow strip of sky immediately above us. Though breadth of vision is essential, Sir Edward Salisbury referred to the underlying truth in the familiar phrase that a little knowledge is a dangerous thing—he would, however, substitute the word "information" for knowledge. In this, of course, he is perfectly correct. "The well informed, but badly educated, as is so often manifest in the political arena, can be of all persons the most ignorant and ill-balanced, with a myopic vision that mistakes the few factors that are apprehended for the whole environment and confuses immediate consequences with ultimate effects." Sir Edward Salisbury then referred to the macroorganisms of higher vegetation and also to the varied flora of microorganisms in our intestinal tracts, and to the astounding capacity for rejuvenation and stability shown by them. He said that a realization of this fact did not justify its abuse, but that it behoved us to learn more of the pattern of life if we were to exercise a control of Nature for the benefit of the human race. He declared that the training which would be received in the laboratories with which he was concerned on that particular occasion would fail lamentably in its main purpose if it did not add materially to the zest of living. In our discussion of twenty years ago, we concluded that if teaching in biology was followed by an elementary course in psychology, the young people of Australia would be better equipped mentally. They would then be sufficiently enlightened to recognize the trend of modern social development and they might also have a share in its direction. If they set themselves to it also, they would be likely to gain some personal satisfaction from what was often the arduous task of living. It is appropriate perhaps to remember the words of St. Paul to the Corinthians: "Though I have . . . all knowledge . . . and have not charity I am nothing."

Current Comment.

DURATION OF ACTIVE IMMUNITY AGAINST TETANTUS.

THE use of tetanus toxoid for active immunization against tetanus is well established, and its effectiveness is not questioned, at least on a short-term basis. Many reports have been published on the immediate effectiveness of this procedure, but there have been few on the question of long-term immunity. In 1951, John A. Bigler published his findings from a ten-year study,[1] the material for his report being a group of 300 infants and children who received primary immunization courses and subsequent booster injections according to various schemes. Over a period of ten years a great many titrations of the children's blood serum for tetanus antitoxin were carried out. It was found that primary or basic immunization could be obtained with either two or three injections of tetanus toxoid. When three injections were given, the third injection acted as a booster injection and produced an effective level of antitoxin in all cases for a period of at least six months. However, when two injections only were used for basic immunization, protective levels were not always attained, and the need was shown for the giving of a booster injection always after an injury, no matter how short the interval since the primary injections. In the long-term study, detectable amounts of tetanus antitoxin were present in the blood serum of all immunized children tested up to a period of ten years, although only in a few cases was this at a protective level. In all cases, however, protective levels of antitoxin were produced after booster injections given up to ten years after immunization. Protective levels were obtained with each of as many as five booster injections. The response to the booster injections was rapid, occurring within the first seven days. It was also found that the greater the number of injections given, the longer the protective levels were maintained. Bigler recommended that until there were further reports on antitoxin levels over long intervals, although apparently protective levels were present, it seemed advisable to give a booster injection to an immunized person at the time of an injury from which tetanus might develop.

A more recent investigation is reported by T. B. Turner, E. S. Stafford and L. Goldman.[2] They set out to examine the status of immunized veterans and children who had been given combined diphtheria and tetanus toxoid and to answer the questions whether the protection was permanent, whether booster doses should be given and when, and whether additional knowledge would render the often used dose of tetanus antitoxin unnecessary. Two groups of subjects were studied: the members of one group had received injections and one booster dose in the armed forces from five to eleven years previously; the members of the second group had received a similar course, but within five years. A control group consisted of 30 persons who had never received tetanus toxoid. The first step in the investigation was to take "pre-booster" samples of serum from all those participating; then a single dose of fluid toxoid (0·5 millilitre) was given into the deltoid muscles, and "post-booster" samples were taken on the fourth, seventh and fourteenth days. The antitoxin con-

[1] *Am. J. Dis. Child.*, February, 1951.
[2] *Bull. Johns Hopkins Hosp.*, April, 1954.

tent of the serum was estimated by using protection tests in mice, the serum being mixed with a dose of toxin known to contain ten minimum lethal doses; toxin controls and controls treated with a standard antitoxin were included. In the first group (that is, those who had received injections and one booster dose from five to eleven years previously) the serum of 45 out of 72 contained 0·1 unit or more per millilitre, and only six had less than 0·01 unit (commonly accepted figures for a protective level of tetanus antitoxin in the blood serum are from 0·01 to 0·1 unit per millilitre). In the second group (that is, those immunized within a period of five years) the serum of 64 of 73 had 0·1 millilitre or more, and in only three cases was it less than 0·01 unit. Seven days after the giving of the booster dose, 54 members of the first group who were retested showed values of 0·5 unit per millilitre of serum, the values extending up to ten units per millilitre. The figures for the second group also showed substantial rises in all cases. The persons who had never previously received toxoid had no detectable antitoxin in their serum; and when they were tested after fourteen days, there was still little being produced. Turner, Stafford and Goldman also made a series of observations on patients receiving 1500 units of antitoxin for treatment, and found that intramuscular injection of antitoxin was not followed by the development of high levels of circulatory antitoxin for some hours at least; the highest figure recorded after sixty hours was 0·25 unit per millilitre. From their investigation of the use of toxoid they conclude that once basic immunization has been established, stimulating doses should not be necessary at intervals closer than five years. Adoption of this procedure would obliterate the dangers of the use of horse serum. Reference to these dangers, of course, brings up another problem altogether, and it might be mentioned that Turner, Stafford and Goldman discuss the possibility of using human serum for prophylaxis in view of the high titres which can be obtained. The idea is of interest, but it might with justification be received a trifle coldly by those responsible for maintaining the supply of human serum for other purposes.

OXYGEN THERAPY AND RETROLENTAL FIBROPLASIA.

THE role of oxygen in the genesis of retrolental fibroplasia was discussed in these columns on March 21, 1953, and again on March 27, 1954. Attention was drawn to reports from England, America and Australia (a paper on the subject by Kate Campbell, of Melbourne, was perhaps the earliest significant contribution in this field) in which an association had been noted between the occurrence of retrolental fibroplasia and the administration of oxygen in high concentration to premature infants. The exact nature of the association was not clear, and it could not be said that a direct cause and effect relationship had been established between oxygen therapy and retrolental fibroplasia, but the advisability of the routine use of prolonged high oxygen concentrations in the nursery was called seriously into question. Certainly, the experimental evidence available indicates that high concentrations of oxygen can cause obliteration of retinal vessels, and this may be the underlying cause of retrolental fibroplasia.

The clinical evidence for a causal relationship receives strong support from a study carried out at the Bellevue Hospital Premature Nursery in New York by J. T. Lanman, L. P. Guy and J. Dancis.[1] In this study, premature infants allotted by random selection were treated with oxygen in respectively high and low concentrations. Eighty-six infants were admitted to the study; 21 died before the age of eight months. Trace was lost of one infant, who was then excluded from the study. Of the remaining 64 infants, 36 received oxygen in high concentrations and 28 in low concentrations. The high oxygen concentration was continued for a minimum of two weeks or until the infant reached a weight of 3·3 pounds. The average of the individual infant's mean oxygen concentration was 69%, with a standard deviation of 6·5%; discontinuation of therapy was abrupt. This treatment, it should be noted, approximated that usually given in the nursery before this study began. Infants in the group with low oxygen concentration received oxygen therapy only for cyanosis. They were then given oxygen from specially prepared tanks containing a mixture of 50 $\pm$ 5% oxygen and 50 $\mp$ 5% nitrogen. The average of the mean concentrations of oxygen was 38% with a standard deviation of 7·7%. Oxygen therapy was discontinued at least once daily and not resumed except on reappearance of cyanosis.

Of the 36 infants receiving oxygen in high concentration, eight developed severe bilateral irreversible cicatricial retrolental fibroplasia. Six of these infants are believed to have no useful vision; two may have useful vision in one eye. Of the 28 infants receiving oxygen in low concentration, none developed retrolental fibroplasia. This difference is statistically significant. The vascular stage of retrolental fibroplasia was seen in 61% of the infants in the group receiving high oxygen concentrations and in 7% of infants in the low oxygen concentration groups. This difference is highly significant. The difference between the mortality rates of the infants in the respective groups is not significant.

Lanman, Guy and Dancis discuss the findings of other investigators, including those of Szewczyk, to which we referred in our previous discussion, and which he interpreted as not confirming the view that high oxygen concentrations were harmful to the premature infant. There are certainly confusing factors in the picture still, and it is possible that many agents are capable of producing retrolental fibroplasia. This, as Lanman, Guy and Dancis point out, may account for the occasional cases occurring in infants who have never received oxygen therapy. However, the clinical disease is almost always associated with the use of oxygen, and the hazards associated with oxygen must be weighed against its possible benefits. The administration of oxygen to premature infants has become something in the nature of a habit, especially in high concentrations, although this habit receives surprisingly little support from a critical examination of the respective mortality rates of infants receiving high and low concentrations of oxygen. Lanman, Guy and Dancis rightly consider it unlikely that the clinician will be much influenced by this negative evidence when faced with a cyanotic infant demanding treatment, and they state that it may be that the unproved benefits of oxygen therapy can be gained without undue hazards of producing retrolental fibroplasia. In their own unit at the Bellevue Hospital oxygen therapy is given only to definitely cyanotic infants and then at measured concentrations of below 40%. Frequent attempts to discontinue therapy are made and it is not resumed except for recurrence of cyanosis; treatment usually lasts from a few minutes to a few hours. Aerosol therapy is given to non-cyanotic infants with compressed air. Under this policy, very little oxygen is used, even for the smallest infants.

Of a different character but leading to much the same practical conclusions is an investigation carried out in the premature baby units of two Manchester (England) hospitals by R. M. Forrester, E. Jefferson and W. J. Naunton.[1] A survey was made of the incidence of retrolental fibroplasia during the years from 1947 to 1953 (inclusive), and this was correlated with the hospital practice as it related to oxygen therapy for premature infants. The results are striking, especially when set out in diagrammatic form. The total number of cases of retrolental fibroplasia was 83, permanent change having occurred in 36. During 1947 and 1948 only one case was recorded. The main incidence occurred during 1950 and 1951 when there were 32 cases with permanent changes (20 with blindness). During 1952 and 1953 only three infants had permanent changes, and none is blind. In both hospitals the rise in incidence corresponded with the introduction of intensive oxygen therapy and the fall

[1] *J.A.M.A.*, May 15, 1954.

[1] *Lancet*, August 7, 1954.

came with its radical modification. Very few infants are now exposed to an oxygen concentration of more than 50%, and the actual duration of exposure has been greatly reduced. Various precautions are carefully followed; their description in this paper will be of interest to those concerned with the practical side of the question. It may be noted that, as in the investigation by Lanman, Guy and Dancis, the Manchester study provided no evidence that restriction of oxygen therapy decreased the survival rate.

Taken by themselves, the findings of Forrester, Jefferson and Naunton could be regarded as having only a *post hoc ergo propter hoc* value; but taken in conjunction with the findings in the controlled study of Lanman, Guy and Dancis and in earlier investigations, they must be seriously heeded. The evidence combines to show that the occurrence of retrolental fibroplasia is directly related to the excessive administration of oxygen and can be controlled by severely limiting oxygen therapy to premature infants. The point scarcely needs further corroboration. No doubt there is room for investigation of other aspects of the problem, and more will undoubtedly be learnt from laboratory investigations of the effect of oxygen. Further clinical investigations along the lines of those carried out by Lanman, Guy and Dancis would, however, be difficult to justify. In the light of the results recorded here, it would be accepting a heavy moral responsibility to expose premature infants to high oxygen concentrations for experimental or any other purposes.

DONOVANOSIS.

NEARLY 50 years ago Major Charles Donovan, who was then a professor and physician in the Madras Medical College and the associated General Hospital, discovered and described the intracellular organisms now known as Donovan bodies which he found in the buccal lesions of patients under his care. The patients were suffering from a condition which has had many names, including *granuloma inguinale* and *granuloma venereum*. Quite recently the name donovanosis has been suggested for the disease, and this has been adopted in a World Health Organization monograph on the subject.[1] The authors, R. V. Rajam and P. N. Rangiah, appropriately enough come from Donovan's old college in Madras, and Rajam is one of Donovan's former students. Donovanosis is not a common condition, but available information on it is rather confused, and the desire to put this in order justifies the preparation of this monograph.

Donovanosis is described here as a chronic, slowly progressive, mildly contagious disease of venereal origin, characterized by granulomatous ulceration of the genitalia and neighbouring sites with little or no tendency to spontaneous healing. It is to be clearly distinguished from *lymphogranuloma venereum*, which is a virus disease affecting the lymphatic systems of the inguino-genito-anorectal regions and is associated with constitutional symptoms. Donovanosis has a fairly wide geographical distribution in both hemispheres. It is endemic in southern China, the East Indies, northern Australia, West and Central Africa, some countries of Central, South and North America, and the West Indies. Epidemics have been reported from South America and New Guinea. The nature of the organisms which Donovan described as the causative agents of the disease has been a matter of controversy. Rajam and Rangiah state that these microorganisms are so constantly present in the lesions of the disease that a clinical diagnosis of donovanosis is not complete without their demonstration, and the disease has been reproduced in human volunteers by injecting a pure substance of Donovan organisms obtained from an unruptured bubo or by transplanting subcutaneously a small piece of granulation tissue from an infected one. Apparently the organism is now regarded as being bacterial and bacillary.

For those who require them, the details of the ætiology, pathology, epidemiology, clinical features, diagnosis and treatment of donovanosis are available in this monograph. Great difficulty was experienced with treatment until the introduction of the antibiotics. The first of these, penicillin, was not effective, and early hopes from the excellent immediate results obtained with streptomycin were somewhat dampened by the realization that the Donovan organism very quickly developed resistance to this antibiotic. However, subsequently developed antibiotics, including "Aureomycin", chloramphenicol and oxytetracycline, have been found to be effective, and the problem of treatment is apparently under control.

In their conclusion to the monograph Rajam and Rangiah state that it is true that the hospital incidence of donovanosis is negligible in comparison with that of syphilis or gonorrhœa in southern India, and that the disease is not a major public health problem. It is, however, a signal example of the close interrelation between poverty and disease, and it seems right that it should have claimed the attention of such a body as the World Health Organization. Donovanosis is described as a terrible affliction which slowly and inexorably ravages its victim. Poorly understood and poorly handled, it becomes so loathsome that the patient is shunned like a leper. There are sanatoria for the tuberculous and leprosaria for lepers, but the patient with donovanosis goes from hospital to hospital for admission. Few general practitioners are interested in the disease. Its victims come from the poverty-stricken under-privileged sections of the population, with poor personal hygiene and debased sexual standards; although, thanks to the low infectivity of the organism, the disease is prevalent only in a small percentage of these sections of the population. In view of the limited nature of the incidence of donovanosis, it should be possible to control and eradicate the reservoirs of infection by organized public health drives. Early diagnosis and adequate therapy with the antibiotics appear to provide the answers to the problem of control.

[1] "Donovanosis (Granuloma Inguinale, Granuloma Venereum)", by R. V. Rajam and P. N. Rangiah; World Health Organization Monograph Series No. 24; 1954. Geneva: World Health Organization. 9½" × 6¾", pp. 72, with 38 illustrations. Price: 10s.

THE CHEMOTHERAPY OF ORTHOPÆDIC TUBERCULOSIS.

CHEMOTHERAPY has made a considerable impression on orthopædic practice, and not least in the management of tuberculosis. For a number of reasons the importance of using chemotherapeutic agents rationally cannot be too strongly emphasized in this field. This particularly relates to their use in combination in the treatment of tuberculosis. The synergistic effect of certain combinations is recognized and must be borne in mind, but so also must the possibility of antagonism. Experimental and clinical findings agree that such antagonism exists, though it is not possible to lay down comprehensive and rigid rules about it. E. Jawetz and J. B. Gunnison[1] have divided the commoner antibiotics into two groups: group I consists of penicillin, streptomycin, bacitracin and neomycin; group II consists of "Aureomycin", chloramphenicol and "Terramycin". They state that members of group I are frequently synergistic with each other, occasionally indifferent, but never antagonistic. Members of group II are neither synergistic with, nor antagonistic to, each other, but simple additive effects are often observed. Sulphonamides may fit in with members of group II. When a member of group I is added to one of group II, the combined result is a function of the relative susceptibility of the microbial strain to the drug in group I; it may be synergism, but it may also be antagonism. It is not possible, however, to label certain drug pairs as "synergistic" or "antagonistic" in every circumstance.

Other investigators have described the members of groups I and II respectively as primarily bactericidal and primarily bacteriostatic. This view is adopted in a paper

[1] *J.A.M.A.*, October 18, 1952.

on the chemotherapy of orthopædic tuberculosis by F. Harwood Stevenson,[1] who adds isoniazid to group I and PAS to group II. He states that the bactericidal antibiotic (or other chemotherapeutic agent), which acts upon actively dividing organisms, may be prevented from so doing by the bacteriostatic agent inhibiting cell division. Thus "Aureomycin" or chloramphenicol may diminish the effect of penicillin; "Terramycin" has been shown to diminish the effect of isoniazid against tuberculosis. Streptomycin, on the other hand, may help penicillin to cure staphylococcal infection by preventing the emergence of penicillin-resistant staphylococci. It may therefore be necessary, if streptomycin and isoniazid are being used, and another antibiotic is proposed to be used against a secondary invader, to test in the laboratory the effect of a combination of the drugs upon the secondary infecting organism. The practical importance of this is brought into focus by Stevenson's assertion that no antibiotic for tuberculosis should ever be given alone. He states that streptomycin in doses of one gramme daily and isoniazid in doses of four milligrammes per kilogram of body weight daily form the best chemotherapeutic combination at present available. Since many patients will require chemotherapy for more than three months, and since the above-mentioned combination cannot be guaranteed to prevent development of resistance beyond that time, it is at present better always to use PAS in a dosage of at least 15 grammes per day with the other two drugs. The antibiotic treatment of secondary infection may require detailed laboratory investigations if antagonism between drugs is to be avoided.

Turning more specifically to the question of orthopædic tuberculosis, Stevenson states that patients admitted to hospital with reactivation of the disease frequently give a history of injury. An instruction to patients after discharge from hospital to report any reasonably serious injury, so that they may receive a timely course of chemotherapy, may prevent or diminish such relapses. With regard to the treatment of persistent sinus, it should be possible to obtain healing by chemotherapy in over 90% of cases if surgery is employed in a proportion of them—namely, those in which surgery is demanded on radiological grounds and those in which evidence of healing does not show within two months of intramuscular streptomycin therapy. From a comparative study of results obtained with and without chemotherapy Stevenson found that with the use of streptomycin the average duration of conservative treatment, before the beginning of reossification, was reduced from fifteen months to seven months, and that approximately 40% of patients with intraarticular hip disease regained a range of movement not seen in the pre-streptomycin series. In both series good results were obtained in cases of extraarticular hip disease, and the difference was therefore probably less significant. The results with synovial tuberculosis of the knee were much better with streptomycin treatment than those ordinarily expected in a similar consecutive series without streptomycin. Stevenson states that, in the opinion of many recent writers, chemotherapy has its greatest value in restoring to tuberculosis-inoculated tissues their powers of healing and thereby allowing tuberculous bone and joint disease to be tackled upon the ordinary principles of surgery. He concludes that streptomycin and the newer antibiotics have already belied the pessimistic agnosticism of 1947. In certain instances, notably in disease of the knee and hip, and in some cases in which there are draining sinuses, it appears that these agents are sufficient to produce a degree of quiescence which may amount to cure.

POLYPI OF THE RECTUM AND COLON IN CHILDREN.

ALTHOUGH polypi of the rectum and colon are not rare in children, few series of cases have been reported. This attaches extra interest to a series of 70 cases reported from experience in private practice by J. Wade Harris.[2] Discussing symptomatology, Harris points out that the chief symptom of polypi in children is painless bleeding; most frequently the blood is present as streaks in mucus, and bloody mucus found in these circumstances in children without some type of acute intestinal condition is almost pathognomonic of polypi. The next most common symptom is prolapse of a mass at the anus; this mass is dark red and may or may not bleed. The third symptom in order of frequency is cramping in the abdomen or in the rectum. This symptom is described as being brought about by Nature's effort to expel the polypus and traction on the bowel wall from intestinal peristalsis. Cramping is a more constant symptom if the polypus is in the sigmoid colon or above, and the amount of cramping is more with the pedunculated than with the sessile polypus. Examination of children for rectal and colonic conditions such as this can present difficulties, and Harris offers a number of practical comments. He states that the first examination should be digital, then proctoscopic. He advises against using a child's proctoscope, averring that in the absence of any congenital abnormality, a regular five-eighths of an inch proctoscope can be used without any difficulty. X-ray study of the colon for polypi is most important for polypi above the level of the proctoscope. Cleaning out of the bowel with a dose of castor oil (Harris advises three ounces for the child three or four years old) followed by enemas is essential. Use of double contrast films is necessary, and the examination takes time, care and patience. In this series the greatest number of polypi was observed in children between the ages of one and six years. Fifty-eight patients had polypi in the rectum, 16 in the sigmoid colon and six above the sigmoid colon. Fifty-two patients had single polypi, seven had two polypi, four had three, and three had four or more. It should be noted that this series did not include any patients with the familial type of polyposis.

The question of the correct treatment is still a matter for debate. Twelve patients in the series required laparotomy with colotomy for removal of the polypi. In eight cases in this series self-amputation of the polypus occurred. For any polypus below the peritoneal fold, Harris uses the snare, but for those above the fold he uses direct fulguration. He points out that in children the occurrence of malignant disease from a polypus is extremely rare. Of a series of questions with which Harris ends his article we may quote two: "How long should the polypus be observed when it occurs above the level of the proctoscope and would require laparotomy?" "At what age should it be attacked?" These questions are, as Harris states, not yet finally answered; but informed surgical opinion would support the view that if a child has been presented for opinion because of symptoms, and X-ray examination reveals a polypus, the surgeon should without waiting perform laparotomy and colotomy and examine the large bowel for the presence of any other polypi.

NEURAL TOXIC EFFECTS OF ISONIAZID.

NEURAL toxic effects must be borne in mind as a potential hazard of isoniazid therapy. G. D. Gammon, F. W. Burge and G. King[3] found signs of peripheral neuropathic changes in 46 of 408 subjects of advanced pulmonary tuberculosis treated with isoniazid. The effects consisted of tingling and painful paræsthesia of the feet and legs, followed by weakness or paralysis, with loss of deep reflexes, first in the ankles, then in the knees. The hands were affected in a few cases. The process was subacute and subsided at once on withdrawal of the drug, unless it had progressed to a severe stage, when weeks were required for even partial recovery. The dosage responsible varied from 4·5 to 13·5 milligrammes per kilogram of body weight. This places a limitation on therapy with higher doses of isoniazid.

[1] *J. Bone & Joint Surg.*, February, 1954.

[2] *Am. J. Surg.*, November, 1953.

[3] *Arch. Neurol. & Psychiat.*, July, 1953.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Chemical Studies on a Basic Peptide from Thymus.

J. G. HIRSCH AND R. J. DUBOS (*J. Exper. Med.*, January, 1954) have made chemical studies on a basic peptide preparation derived from calf thymus. They describe in detail the preparation of the material from crude thymus and its solubility and stability. Its activity could be destroyed on incubation with trypsin. Qualitative tests showed that it contained cystine, tryptophane, arginine, a benzene nucleus and an amide linkage. A negative Molisch test result indicated absence of carbohydrate, and no phosphorus was present. The substance could not be crystallized, but it behaved as a single strongly basic compound in an electrical field. The most noteworthy finding of the chemical analysis was the absence of sulphur-containing substances.

Infectious Hepatitis Spread.

V. KNIGHT, M. E. DRAKE, E. A. BELDEN, B. J. FRANKLIN, M. ROMER AND L. O. COPPLE (*Am. J. Hyg.*, March, 1954) have studied the characteristics of spread by infectious hepatitis in schools and households in one epidemic in a rural area in Missouri. The disease had been sporadically recorded in the district over some years, but the district nurse reported an increase of cases in January, 1951, and 65 cases had occurred by April 1, when the study was begun. All the 152 patients observed had actual jaundice persisting over varying times up to two months, and there were no fatalities. An illness without jaundice also occurred amongst some of the children, many of whom had enlarged livers and gave abnormal results to the cephalin-cholesterol flocculation test. The schools appeared to be the chief centres of spread of the disease; and once infection was introduced, the homes continued as small intense foci of spread, attack rates in homes being higher than in school. A small group of children was treated with a small dose of γ globulin (0·01 millilitre per pound of body weight), but no useful information was gained, as the control groups showed a very low incidence of jaundice during the period of observation.

Antibiotics and Coccal Infections in Mice.

J. L. GOWANS (*Brit. J. Exper. Path.*, April, 1953) has continued his studies on the effect of some antibiotics on coccal infections in mice. He attempted to assess the relationship of phagocytosis to the infecting bacteria by comparing the effect of antibiotic treatment on two groups of mice, one rendered very granulopenic by a single subcutaneous injection of three milligrammes of 1:4 dimethanesulphonosybutane. The mice were injected intraperitoneally with virulent organisms, and individuals were sacrificed at varying periods of time, films being made from the peritoneal exudate to study the cellular reaction, and cultures to assess the effect of the antibiotic on the viability of the organism. Among the normal mice, untreated animals died within thirty-six hours; animals treated with either penicillin or "Terramycin" survived up to ninety hours. The cultures from the treated animals showed a fall in the number of organisms in three hours, after which it steadily rose. Examination of the smears showed that phagocytosis was taking place in three hours, and macrophages were more actively phagocytic than polymorphonuclear cells; but by the end of twenty-four hours the cells were degenerating and releasing cocci. In the leucopenic mice the fall in the numbers of living organisms does not take place, and the cells do not exhibit much active phagocytosis. It was thought that in the experiments in which pneumococci were used, lysis of organisms took place in the exudate, but that in experiments with streptococci, the polymorphonuclear cells actually assisted the antibiotic in killing the organism.

Destruction of Bacterial Virus.

S. L. CHANG, P. C. G. ISAAC AND N. BAINE (*Am. J. Hyg.*, May, 1953) have studied the dynamics of the removal of bacterial virus in water by flocculation with aluminium sulphate. This bacteriophage acted upon *Micrococcus pyogenes albus* as in earlier studies. The authors found that increasing the concentration of alum from 40 to 100 parts per million steadily increased the percentage removal of virus in the supernatant of the treated water. Increases in pH value from 5·2 to 8·2 decreased the removal of virus from water treated with 60 parts per million of aluminium sulphate. The optimum pH in relation to the phage used was 5·2. The process of flocculation for removal of virus from water appears to have two stages: first an aluminium virus precipitate, and then aggregation of the precipitate to form flocs; the first is a protein-salt production, the second probably a complex colloidal change. At pH 7·6, 60% of the phage was reactivated after a period of twenty-one hours. The authors point out that one result of the treatment of water is a concentration of virus in the floccules, and not its destruction.

HYGIENE.

Ozone Toxicity.

H. E. STOKINGER (*Arch. Indust. Hyg.*, May, 1954) has reviewed published reports on the biological effects of ozone under the headings of properties, occurrence and preparation, toxicity, uses, human exposure and sources. He presents the following facts and impressions as a summary of these reports. Ozone as prepared with the usual precautions to minimize impurities, such as nitrogen oxides and other oxidants, is both acutely and chronically toxic. The chief injurious effect of ozone inhalation is on the lungs. This consists of pulmonary œdema and hæmorrhage, which if sufficiently severe may cause death within three to forty-eight hours; if less severe, it may cause temporary pulmonary embarrassment, from which the animal recovers without sequelæ. No instance of death from exposure to ozone in man has been reported. Secondary effects consist of irritation of the mucous membranes, headache and drowsiness. Low concentrations have a narcotizing and general depressant effect, producing sleep in certain individuals as has been observed in animals. Approximately one-fourth of the atmospheric ozone concentration reaches the pulmonary vessels, three times as much being destroyed in the upper respiratory passages, according to deductions from the rate of development of effects in tracheotomized animals compared with those breathing normally through the nose. No ozone has ever been detected in expired air. Ozone appears to cause a decreased rate of pulmonary ventilation and lowered output of carbon dioxide. No toxic substances have been demonstrated in the blood of animals inhaling ozone. There are insufficient data to show conclusively that ozone prepared under usual conditions is appreciably more toxic than pure ozone. Impurities consist of varying amounts of oxides of nitrogen. Other impurities may be present whose toxicities are unknown. Whether the toxicity of ozone is appreciably altered by whatever small amounts of impurities accompany ozone as it is commonly prepared has yet to be demonstrated. Reports of occupational illnesses believed to have arisen from ozone exposure have lately appeared, and it would seem that ozone-generating sources should be held suspect as potential sources of human injury. The opportunities for ozone exposure are increasing. Although this is chiefly due to increasing use of electrically powered equipment, ozone is also finding increasing applications in the chemical industry, in treating industrial wastes and in preserving perishable foods.

Summer Topical Fluoride Programme.

F. WERTHEIMER (*Am. J. Pub. Health*, April, 1954) gives details of a plan to use dental undergraduates during vacation months in a dental hygiene programme to apply fluoride to the teeth of school children, under the supervision of the dental section of the Michigan Department of Health. This is additional to the routine dental work of the department. In the plan, the local community has the following responsibilities: to inform the community of benefits of treatment; to determine the potential case load; to make decisions on such matters as the age groups to be included, fees to be charged, salaries to be paid and ways of handling indigency; to send out consent cards; to collect moneys and pay expenses and salaries; to order equipment and supplies; to locate housing for students; to hire assistants or arrange for volunteers; to arrange with local dentists for supervision of students; to prepare schedules and send out notices of appointments. These things are usually done by local committees, which may consist of representatives from local dentists, local health authorities, local schools, parents' associations and various other local organizations. The dental section of the central authority assumes the following responsibilities: to produce a consultation service to local committees; to provide pamphlets and talks, and to assist in the preparation of

publicity material; to procure student operators and a permit from the Michigan State Board of Dentistry; to plan and conduct an orientation course; to assume general supervision; to lend portable dental equipment; to prepare master schedules and assist with preparation of the final schedule; to provide necessary record cards and similar material; to evaluate programmes; to assist with ordering of equipment and supplies; to help with selection of a treatment room. The unit usually consists of an undergraduate dentist, an assistant who acts as clerk, and one or more part-time voluntary assistants. The author states that the students who participate receive the following benefits. They have experience in the group method of topical fluoride application. They develop skill in handling children. They learn about dental conditions of children in communities, in contrast to the select group usually seen in dental school. They obtain orientation in public health and the concept that dental disease is a public health problem, as well as the opportunity to observe community action as an approach to the solution of a problem. It has been found that child participation is not as great as during the school year, and transportation sometimes creates a problem. On the other hand, adequate space is readily obtainable in the school buildings, and the available supply of student personnel enables a service to be rendered that would not be possible otherwise.

Asphyxiation in Street Manholes.

G. S. MICHAELSON AND W. E. PARK (*Pub. Health Rep.*, January, 1954) report the results of investigations carried out to find the reason for the death of a workman in a water-main manhole. The manhole was four feet in diameter and nine feet deep; it was constructed of concrete bricks sealed with mortar and had a standard iron cover at street level. It was situated in a low swamp area. Men had entered the manhole on the morning of the accident and twice daily for the two previous weeks. Six days after the accident samples of air contained no combustibles and had no odour. The oxygen content was 16%. Two weeks later the oxygen content had fallen to less than 3%. The hole was blown out with fresh air, and the oxygen content fell from 18% to less than 2% in about three hours. The oxygen demand of oil-sand-rock taken from the manhole was found to be three times that of black garden soil and infinitely greater than that of clean sand. Conditions in other similar manholes were then studied. Many were deficient in oxygen. The air in another manhole was displaced with nitrogen and the manhole sealed. The oxygen content of the air in the manhole was then found to rise gradually from nil to 19% in thirty-six hours. The authors state that this series of experiments emphasizes the necessity for caution. No manhole should be considered safe until it has in some way been demonstrated to contain sufficient oxygen or has by some mechanical means been freshly blown out with fresh air. Changes in barometric pressure are relatively minor factors in altering the oxygen content of air in manholes, and the presence of organic matter and other extraneous materials which have a chemical oxygen demand is a factor of major importance. Four specific conclusions, however, were reached: manholes, particularly those in low or swampy areas, are potentially dangerous in respect to oxygen depletion; there may be substances in the subsoil other than vegetable matter which may increase the chemical oxygen demand; there is free and rapid diffusion of gases through the walls and floor of manholes; the flow of air through the manhole cover openings is ineffective under all conditions in maintaining the air within the manhole in equilibrium with the outside air.

Food and Food-Handler Sanitation.

E. RUSSELL JACKSON (*Am. J. Pub. Health*, May, 1954) discusses effective food service sanitation. He states that the adoption of uniform clear and simple requirements is essential. These should have the approval and support of the food service industry. A local board of review, consisting of representatives from the industry, from the official control agency and from the consumer group, should assist with the problems that may arise and differences that may occur between industry and the controlling agency. Local authorities should be informed at special meetings of the reasons and facts that support the need for food service sanitation by means of graphs, charts and pictures of existing conditions. A qualified representative of the central health authority should be at local authority meetings when the proposed requirements are being discussed. Food service personnel should be educated in the principles and practices which will enable them to prepare, store and serve food safely, and they should be taught an appreciation of basic personal hygiene. In addition, the public should be educated in food sanitation to enable them to demand a high standard of sanitation in the food industry as well as a high standard in their own homes. Surveys of standards of food sanitation by central health authorities assist by providing estimations of the degree of attainment of adopted standards. They also indicate weaknesses in enforcement of the required standards which may be given additional attention. The author considers that food sanitation can be further advanced by the use of devices such as slogans, placards, flow charts, utensil swab tests and bacteria growth demonstrations. Incentives and forms of recognition may be developed to encourage high sanitary standards.

Polonium in Urine of Miners Exposed to Radon.

M. SULTZER AND J. B. HURSH (*Arch. Indust. Hyg.*, February, 1954) have measured the concentration of a radon decay product, Po^{210}, in the urine of miners exposed to radon concentrations in the air. These measurements were made to explore the possibility of using them to estimate the amount of previous exposure to radon of workers exposed to hazards associated with uranium. It was found that concentrations of Po^{210}, varying from about two to 38 micromicrocuries per litre, were present in the urine of miners working in the uranium mines of the Colorado Plateau. In the case of laboratory personnel used as subjects, no urinary polonium could be detected. The polonium in the miners' urine is belived to come from Pb^{210} stored in the bone, which, in turn, is derived about equally from radon daughter products deposited in the lung and radon accumulations in the body fat (10 kilograms of fat). The authors suggest that urinary polonium measurements may provide a method of estimating the integrated exposure of the lungs to radon and its daughters. They have shown that, even though body burdens of radium equal to the maximal permissible level were accumulated by uranium miners, this amount of radium would contribute a negligible amount of polonium in the urine. With the use of probable values for the fate in the body of the chemical elements in the radon decay scheme, the miners' daily urinary polonium output was calculated and found to be within the range of the reported experimental values. The calculations are included as appendices.

Dermatoses Among Office Workers.

G. E. MORRIS (*J.A.M.A.*, July 25, 1953) states that skin eruptions among office workers are not common. Among 1213 patients treated by the author for industrial dermatitis 36 were office workers, who contracted the conditions as follows: four from carbon paper, five from paper, six from glue, two from cardboard, twelve from various machine dyes and inks and the chemicals used to remove them, and one from paper towels; in addition six cases described in this report were of special interest. In the first of these the patient was proved to be allergic to an elastic rubber band, to a green rubber blotter, to a green leatherette chair and also to a red carbon paper, positive patch test results being obtained with all these. Three office workers were treated for eruptions emanating from their use of "rubber thumbs" or rubber finger tops while counting or picking up cards. The author suggests that when an office worker complains of a rash that he believes to be due to something encountered in his work, it is only prudent to test him with the various materials with which he comes in daily contact.

Test for Blood Cholinesterase Activity.

J. H. WALPSIE AND G. D. WINTER (*Arch. Indust. Hyg.*, May, 1954) report in-vivo and in-vitro studies to correlate the end-point colours of the Limperos and Ranta BTB test with different degrees of blood cholinesterase activity, showing values ranging from near nil to 100% activity. They state that especially if one regards these colours as representing a "range" of cholinesterase activity, rather than a fixed percentage of cholinesterase activity, then one has in this test a valuable tool for quickly and easily screening persons who may have been exposed to a cholinesterase inhibitor. The actual method is simple and rapid; the equipment and reagents required are few and, in America, inexpensive and easily obtained. The organic phosphate insecticides are cholinesterase inhibitors, and their toxic effects result from this property. Persons preparing or using these insecticides can be easily screened for early toxic effects by means of this test. Any with lowered cholinesterase activity should be removed from further possible exposure and checked by the more accurate Michel electrometric method. If toxic effects are confirmed, the cause of the exposure should be investigated and removed and action taken to prevent recurrences.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

CVII.

THE CARE OF THE PREMATURE INFANT.

DEFINITION.

THE International Committee at Geneva in 1937 defined as a premature infant any baby who weighed five and a half pounds or less at birth. While this is a simple and convenient definition, it is far from being accurate. Many full-time infants may have this birth weight, and in the British survey of 1946 only 71% of infants weighing five and a half pounds or less at birth were premature. It is probably advantageous for these small full-time infants to have the special care accorded to premature infants, provided they are not kept too hot.

On the other hand, infants of indubitable prematurity may be over five and a half pounds in weight at birth, and their well-being is jeopardized if they are treated as full-time babies. It is therefore important to diagnose prematurity in these infants at birth.

The most helpful signs are the ill-developed soft cartilage in the ears, the absence of mammary tissue, the low hair-line on the forehead extending down into "sideboards" over the cheeks, the fine skin with lanugo, the ill-developed toenails, and in females the ill-developed *labia majora* with very prominent clitoris and *labia minora*.

PRENATAL AND NATAL CARE.

Prenatal Factors.

The fate of the premature infant is very greatly influenced by prenatal factors, of which the most important is the gestational age of the fœtus. The survival rate is very low in fœtuses of under twenty-eight weeks' gestation. It rises sharply after thirty-two weeks and still more sharply after thirty-four weeks. Infants of over thirty-four weeks' gestation (birth weight four pounds six ounces) usually have a good prognosis. The most recent figures from good centres in the United States of America are shown in Table I.

TABLE I.

Period of Gestation.	Birth Weight.	Mortality. (Percentage.)
Up to 28 weeks	2 pounds 3 ounces or less (1000 grammes or less).	90–100
28 to 32 weeks ..	2 pounds 3 ounces to 3 pounds 5 ounces (1000 to 1500 grammes).	45– 55
32 to 34 weeks ..	3 pounds 5 ounces to 4 pounds 7 ounces (1500 to 2000 grammes).	5– 15
34 to 36 weeks ..	4 pounds 7 ounces to 5 pounds 8 ounces (2000 to 2500 grammes).	4– 6

Other prenatal factors are the mother's health during pregnancy, the cause of the prematurity and the type of delivery. It is important that the infant be delivered in an institution where premature babies can be adequately nursed. Moving the infant is prejudicial to it. Therefore it is better to transfer the mother before birth, rather than to transport the baby after birth.

Preparation for Birth of Premature Infants.

At the birth of the premature infant everything should be in readiness. The following items are required:

1. A lined cot warmed to a temperature of 90° to 95° F., which should be at the bedside or in the theatre. If the cot is heated by bags, three are usually required, one at either side and one at the foot. There should be three thicknesses of blanket between the baby and the hot water bottle cover. Occasionally an extra hot water bag may be necessary, and one under the mattress is useful in warming the cot. A bath thermometer placed in the position in the cot which the infant will occupy is a useful aid in maintaining the correct cot temperature.

2. A sterile mucus catheter with a glass trap.

3. An oxygen cylinder with a suitable connexion and a sterile intranasal catheter (size 3). A spare cylinder should always be at hand also.

4. A bowl of sterile water to test the rate of oxygen flow.

5. Ampoules of chemical stimulants, such as "Lobeline" and nikethamide ("Coramine", "Anacardone").

6. Ampoules of vitamin *K* analogue.

7. A sterile wrap weighed and warmed. If cotton wool is used, it must be covered with gauze.

8. A sterile pad for the umbilical cord.

The Birth.

The care of the premature infant begins with the onset of labour, and the fate of the baby is often determined at birth. During parturition there are two main dangers: (*a*) hypoxæmia and (*b*) intracranial trauma.

The prevention of fœtal hypoxæmia and consequent deep intrauterine respiration is very important, particularly if we accept the view of the Boston school on the ætiology of hyaline membrane resorption atelectasis. Intracranial trauma is predisposed to by the soft ill-ossified skull, which allows of over-moulding, and the immature development of the walls of the veins. Episiotomy is usually performed. Breech birth is a particular hazard because of the small size of the dilating wedge—the breech—in comparison with the relatively large head.

At birth it has usually been the custom to endeavour to allow the baby to get the placental blood by waiting till the cord has ceased pulsating. The premature infant has a relatively larger placenta as compared with his body weight than has the full-time infant, and therefore his placenta contains a relatively larger amount of blood than that of the full-time infant. It is possible that we do the premature baby a disservice by housing all this extra blood in his circulation and creating a plethora.

At present we make the first priority at birth the sucking out of liquor and mucus before the baby breathes. The premature baby has not the reflexes necessary for the removal of the liquor from the respiratory passages. The infant should be kept with the head low till the sucking out is complete. It is wise also to aspirate the stomach.

Since we have concentrated on this pre-breathing sucking out, the incidence of hyaline membrane atelectasis has decreased. The baby should not be allowed to become cold during this procedure. The sucking out should be done with round-ended catheters and care taken to avoid injuring the buccal, pharyngeal, œsophageal and gastric mucous membranes. Mucus catheters should be stored in sterile containers. After use the end entering the infant's mouth should be carefully placed in a sterile towel or receptacle. Where mouth-suckers are used, the end used by the operator must not be allowed to touch the end entering the baby's mouth, or contamination with the operator's throat and mouth organisms will occur.

In the case of the infant delivered by elective Cæsarean section before the rupture of the membranes, it is particularly necessary to remove the liquor from the upper part of the respiratory tract. In these cases the liquor is present in much larger quantities than occurs in a natural birth. Since adoption of the manœuvre suggested by Dr. June Pash and described by her elsewhere in this issue, the results have been greatly improved. This consists, in a vertex presentation, in sucking out the liquor from the uterus and also aspirating the infant's pharynx while it is still in the uterus before it has been handled at all. In this way we can forestall the first breath and prevent the inhalation of blood and liquor.

If the baby is slow to breathe, oxygen is given by intranasal catheter; and if there is still no response 0·5 millilitre of "Lobeline" is given. If this is not effective, nikethamide can be given in a dose of one minim per pound of estimated body weight.

After the umbilical cord is cut, it is covered with a sterile pad. It is very important to prevent infection of the freshly cut cord. The infant is placed in his sterile wrap and transferred to the warmed cot. He is placed on his side with the head low. If there has been adequate sucking out, it is not necessary to elevate the foot of the cot. This hinders the movement of the diaphragm, which is the important respiratory muscle for the premature infant. If the infant's colour is not satisfactory, he is given oxygen. An intramuscular injection of vitamin *K* analogue is given (1·0 millilitre to a four or five pound baby, 0·5 millilitre to a two or three pound baby).

CARE AFTER BIRTH.

The baby is transferred to the premature nursery.

Where infants are nursed in lined cots the room temperature is kept at 70° F. and the humidity is maintained between 60% and 65%. The great majority of premature

infants are nursed in such cots, and with good nursing the cots are entirely satisfactory. The temperature of the cot is maintained at 90° to 95° F. for the small premature infants and at a lower temperature for the premature infants of greater gestation. As the infant becomes older also the temperature of the cot will need to be lowered. For each baby the temperature of the cot should be that which keeps his groin temperature between 97° and 98° F. The hot water bags are filled in rotation and as often as necessary to maintain the desired temperature. If a bag under the mattress has been necessary, it is advisable to dispense with it as soon as possible, as it entails lifting the baby on his mattress. The more elaborate incubators, humidified, electrically heated and automatically controlled, undoubtedly save nursing time, and the closed type is beneficial in that it ensures isolation of the infant.

Position.

On arrival in the ward the infant is not oiled or handled. He is kept lying on his side with the cot flat and is turned from side to side three-hourly if his condition permits—or less frequently.

Oxygen.

If the baby can maintain a satisfactory colour without oxygen, there is no need to administer it. Since I believe that the excessive use of oxygen in premature infants results in retrolental fibroplasia, I think that it should be given only when necessary therapeutically and not prophylactically.

When it is given, the flow should be kept to that which is just sufficient to maintain a satisfactory colour. If it has been necessary to use continuous oxygen administration for many hours, it should be reduced slowly, the flow being cut by a litre each two hours. Should the baby have required continuous oxygen for some days, the flow is reduced by one litre each four hours. It should never be stopped suddenly. The sudden reduction in oxygen to atmospheric level produces a period of relative hypoxæmia which is potentially dangerous to the infant.

The method of administration depends on the circumstances. It is useless to give oxygen by tent or cot to an infant who has ceased breathing or whose respirations are infrequent and shallow. These infants must have the oxygen introduced into their bodies, and for them oxygen is administered intranasally. Åkerren's method of intragastric administration (Åkerren and Furstenburg, 1950) entails rather much handling for a sick premature infant.

The intranasal catheter size 3 is passed horizontally along the floor of the nose for a distance equal to the measurement from the nostril to a point just anterior to the ear. It will often pass more readily down one nostril than the other. The rate of flow should always be tested prior to passing the catheter. It is usually advised that this should be about five bubbles per second. This can usually be attained with a flow of about one litre per minute.

If the baby is breathing sufficiently well to inhale it, the oxygen can be given by tent or oxygen cot.

All oxygen used for infants should be warmed and humidified.

Any mucus or fluid obstructing the upper airway should be aspirated. If the initial sucking out at birth has been thoroughly done, it is rarely that subsequent aspirations are necessary. Each aspiration is a danger to the baby, as it is a possible means of infection. Indeed, too frequent aspirations may cause pharyngitis with the production of mucus, sometimes blood-stained, necessitating more aspiration, and so a vicious circle is set up.

Temperature.

Taking the temperature rectally exposes the baby to more risk of infection than when it is taken by other routes. Proctitis can also occur from the use of thermometers imperfectly cleansed of antiseptic solution or from the trauma of frequent insertion of unlubricated thermometers.

The axilla is usually more exposed than the rest of the baby and taking temperatures here may give deceptively low readings. We use the groin temperature and leave the thermometer *in situ* for five minutes.

I find it wise to record the temperature hourly for the first six hours and thereafter three-hourly as long as the baby is in the premature ward. We keep it between 97° and 98° F. If the temperature falls too low, the infant becomes lethargic and œdematous. If it is too high, he will become dehydrated.

Antibiotics.

If the membranes have been ruptured for some time, if the infant has required much resuscitation or sucking out, and in cases of atelectasis, it is usual to give a prophylactic course of antibiotics for four days—namely, preparations of procaine penicillin fortified with crystalline penicillin 100,000 units daily to four and five pound infants and 50,000 units daily to two and three pound infants, with streptomycin 20 milligrammes per pound of body weight daily.

When to Handle.

The infants are left undisturbed save for the temperature taking and turning for a period depending on the condition of the individual baby. If the condition is satisfactory, we leave babies of under three pounds for forty-eight hours and babies of over three pounds for twenty-four hours. Babies of five pounds and over may be left for twenty-four hours, or if they appear hungry and vociferous, they may be left for only twelve hours. It is not wise to delay feeding long after the baby cries, as the crying dehydrates him.

Weighing.

The scales should be brought to the side of the cot and the infant weighed in his wraps. If these have not been previously weighed, they can be weighed subsequently. The infant is not oiled.

Feeding.

The first feeding is usually given about two hours after the weighing, provided the condition is satisfactory. Some workers delay all feeding routinely for three days and often longer. The work of Hansen and Smith shows that quite marked dehydration occurs in infants from whom all fluid and food is withheld for seventy-two hours. Clinically the infants appear dehydrated after three days, and I think that there is no doubt that the kernicterus rate is much higher in these dehydrated infants with delayed feeding.

Many premature infants show œdema associated with free urination. Once the œdema goes, their hydration requires watching.

Most infants of under three pounds will commence feeding at the beginning of the third day, and most of over three pounds at the beginning of the second day. Five pound infants may require feeding after twelve hours.

If an infant is too sick to be fed at the end of forty-eight hours and has no œdema, it is wise to institute a subcutaneous drip. (For details see appendix.)

Before feeding, place the infant on his right side and elevate the head of the cot. It is wise to keep it elevated constantly thereafter.

The Mode of Feeding.

The mode of feeding will depend on whether the infant is able to suck or not. This, in turn, depends on the period of gestation rather than the birth weight. Generally speaking, most babies of four pounds and over can suck. Most infants of under four pounds will require gavage, though some three pound infants can take all or part of their feeding from a bottle.

If babies can suck, they can be fed with a bottle. If they cannot suck, they can be fed either with a pipette or by means of gavage. While theoretically pipette-feeding is held to be simpler than gavage, it actually requires more skill. It is often difficult to avoid giving the feed at the time when the baby is inspiring, and the risk of aspiration is present. If a baby cannot suck, therefore, we feed him by gavage.

Gavage.

Many people are afraid that a gavage catheter may pass into the trachea. When the catheter is passed via the mouth I have never known this to occur. The real and ever-present danger with gavage feeding is the possibility of giving too big a feeding which is subsequently regurgitated and aspirated. This may cause suffocation or insufflation bronchopneumonia. The size and composition of the feeding must therefore be carefully controlled and suited to the individual infant.

The technique of gavage is as follows:

The requirements are a sterile catheter, size 3 or 4, a five or ten millilitre syringe, rubber tubing and a glass connexion to allow observation of flow of feeding. The distance from the root of the nose to the xiphisternum is measured in a straight line. This indicates the distance from the lips to the œsophagus just above the cardia. A piece of cotton is tied around the catheter at this point. A second piece of cotton is

tied around the catheter one or one and a half inches further from the tip. When the catheter is passed to this mark, the eye will be in the stomach.

As infants are all different lengths, it is important that each infant have his own individually marked gavage catheter. With surgically clean hands the nurse passes the catheter into the mouth and down the œsophagus till the second mark reaches the lips. She then attaches the syringe and gently aspirates to note if any gastric residue is present. If any residue is present, it is measured. The catheter is then withdrawn so that the first mark is level with the lips. The syringe is disconnected and the piston removed. The barrel is then attached to the catheter to act as a funnel. The catheter is then pinched and the feeding poured into the barrel. Any air present is allowed to escape, and then the feeding is run in. When the feeding has passed the glass, the nurse counts about 12 slowly. She then pinches the catheter at the lips and withdraws with an upward movement. The baby does not swallow air with gavage feeding, and there is no need therefore to eructate his wind afterwards.

Nurses are usually instructed to pinch the catheter when they have first passed it. If the infant becomes cyanosed, it is assumed that the catheter is in the trachea. If the colour remains good it is known to be in the gullet. This manœuvre gives the nurses confidence, but it is doubtful if it has any other value.

If an infant becomes cyanosed or distressed during a gavage feeding, the catheter should be withdrawn immediately.

The First Twenty-Four Hours of Feeding.

It is advisable to give only boiled water for the first feeding in case there is any difficulty. Thereafter 5% glucose in water is given.

The size of the feeding varies with the individual baby. It is generally a safe rule to give at each feeding half as many drachms as the baby weighs in pounds.

Before running in the feeding the gavage catheter is passed into the stomach and the stomach is aspirated for any residue. In this way it can be ascertained if the previous feeding has left the stomach.

Interval and Frequency of Feeding.

The infants are fed three-hourly, eight times daily. After each feed the baby is left lying on his right side for one and a half hours and then turned to his left side for one and a half hours. In this way the infant will always be placed on his right side before the feeding. This diminishes the risk of regurgitation of gastric content through the lax cardia.

TABLE II.
Scheme of Feeding.[1]

Weight of Baby. (Pounds.)	Day of Life.	Strength of Feeding.		Composition of Feeding.		Size of Feeding. (Drachms per Feed.)	Remarks.
		Expressed Breast Milk.	Glucose Solution (5%).	Expressed Breast Milk. (Drachms.)	Glucose Solution (5%). (Drachms.)		
2[2]	1	—	—	—	—	—	
	2	—	—	—	—	—	
	3	—	Whole.	—	1	1	
	4	1 part.	3 parts.	½	1½	2	
	5	1 part.	3 parts.	½	2½	3	
	6	1 part.	2 parts.	1½	2½	4	Increase more slowly if any vomiting, pre-gavage gastric residue or frequent stools. Very premature infants may not tolerate full expressed breast milk till four weeks old.
	7	1 part.	2 parts.	1½	3	4½	
	8	1 part.	1 part.	2½	2½	5	
	9	1 part.	1 part.	2½	2½	5½	
	10	2 parts.	1 part.	3½	2½	6	
	11	2 parts.	1 part.	4	2	6	
	12	3 parts.	1 part.	4½	1½	6	
	13	3 parts.	1 part.	4½	1½	6	
	14	5 parts.	1 part.	5	1	6	
	15	Whole.	—	6	—	6	
3[2]	1	—	—	—	—	—	
	2	—	Whole.	—	1½	1½	
	3	1 part.	3 parts.	½	2½	3	
	4	1 part.	2 parts.	1½	3	4½	
	5	1 part.	1 part.	3	3	6	Increase more slowly if any vomiting, pre-gavage residue or frequent stools.
	6	2 parts.	1 part.	4	2½	6½	
	7	3 parts.	1 part.	5	2	7	
	8	6½ parts.	1 part.	6½	1	7½	
	9	Whole.	—	8	—	8	
	10	Whole.	—	8½	—	8½	
	11	Whole.	—	9	—	9	
4[3]	1	—	—	—	—	—	
	2	—	Whole.	—	2	2	Bottle-fed infants can be fed to appetite if they appear to want more than these amounts and can manage the feeds satisfactorily. If jaundiced, keep longer on 3 parts expressed breast milk and 1 part 5% glucose.
	3	1 part.	1 part.	2	2	4	
	4	2 parts.	1 part.	4	2	6	
	5	3 parts.	1 part.	6	2	8	
	6	Whole.[4]	—	10	—	10	
	7	Whole.	—	12	—	12	
5[3]	1	—	Nil or Whole.	—	2½	2½	Feed at 12 to 24 hours, depending on baby's hunger.
	2	—	Whole.	—	2½	—	Give mixture if baby hungry.
			or		or		
		1 part.	1 part.	1½	1½	2½	
	3	1 part.	1 part.	2½	2½	5	Feed to appetite if baby appears to want more than these amounts.
	4	2 parts.	1 part.	5	2½	7½	
	5	3 parts.	1 part.	7½	2½	10	
	6	Whole.	—	12½	—	12½	
	7	Whole.	—	15	—	15	

[1] Subsequent feeding: When baby reaches five and a half or six pounds, reduce the feeds to seven feeds daily. (Divide the total feed for the day by seven and give this amount at each feeding.) Later give six feeds daily. (Give one-sixth of the total feed for the day at each feed.) Do not merely omit a feeding.

[2] To obtain accurate size of individual feedings for babies of two and a half pounds and over, and for those of three and a half pounds and over, proceed as follows (these are for three-hourly feeds with eight feeds daily): on the first day of feeding, offer an amount per feed equal to one-half drachm for each pound of baby's body weight. On the second day offer twice the amount for the first day. On the third day, offer three times the amount for the first day. On the fourth day, offer four times the amount for the first day. Thereafter increase the size of feeding by one-half drachm each day, or as tolerated up to three drachms per pound of body weight per feed, but do not increase unless feeding is well tolerated. Strength of feeding will be as in the scheme given above for feedings of two pound and three pound babies respectively.

[3] To obtain accurate size per feeding for babies who weigh four and a half pounds and over, and for those five and a half pounds, calculate as follows (three-hourly feed eight times daily): On the first day of feeding, offer an amount per feed equal to half a drachm per pound of body weight. On the second day, offer twice the amount for the first day. On the third day, offer three times the amount for the first day. On the fourth day, offer four times the amount for the first day. On the fifth day, offer five times the amount for the first day. On the sixth day, offer six times the amount for the first day, that is, an amount equal to three drachms per pound of body weight. Increase feeding more slowly if not well tolerated or if baby does not appear to want this amount. Strength of feeding will be as in the scheme given for babies of four pounds and five pounds respectively.

Type of Feeding.

It is particularly important that premature infants should be given breast milk feeding in the first two weeks. (For method of sterilizing expressed breast milk see appendix.)

It cannot be too strongly stressed that each infant must be assessed individually, and the feeding must be adapted to each baby. The ability to manage increases in strength and size of feedings depends more on the period of gestation than on birth weight.

An infant who is heavy for his period of gestation will need to advance more slowly than the scheme given below, and one who is light for his period of gestation may be hungry and may need to advance more rapidly.

It is generally advisable with the smaller premature babies not to increase the strength of the feeding till twelve hours after increasing the size of the feed.

If there is any vomiting or pre-gavage gastric residue, give 5% glucose solution for the next one or two feedings and then return to the feeding which was managed satisfactorily. Thereafter increase the strength more slowly than in the scheme given.

Any scheme of feeding giving definite amounts must be used as a guide only and must be adapted to the individual infant. The scheme given below is reasonably safe.

The infants requiring gavage must be very carefully watched. With them there are two dangers: (*a*) Overdistending the stomach with big feedings causing regurgitation or vomiting and (*b*) giving insufficient fluid with the risk of dehydration and so increasing the jaundice. Infants who are strong enough to suck can usually take the amounts set out without trouble and, indeed, in the early days often take considerably larger amounts.

The basis of the scheme is to give the infant on his first day of feeding at each feed half as many drachms as he weighs in pounds; this gives during the day half as many ounces as he weighs in pounds.

This amount is doubled on the second day of feeding, trebled on the third day and quadrupled on the fourth day, so that the infant is then receiving at each feed two drachms per pound of body weight—that is, two ounces of fluid per pound of body weight per day. Thereafter the feeding is increased more slowly, half a drachm per feed each day (that is, half an ounce per day) till the baby receives per feed three drachms per pound of body weight (that is, three ounces per pound of body weight daily), which is the full fluid requirement.

On the first day of feeding the baby is given 5% glucose solution and thereafter diluted breast milk. Infants of two and three pounds are started with one part of expressed breast milk and three parts of 5% glucose solution (9·5 Calories per ounce). The two pound baby stays on this for two days and the three pound baby, provided he is managing it, for only one day.

The strength of the feeding is then increased, in the case of the two pound baby, every other day, and for the three pound baby every day. This increase in strength is entirely contingent on the baby's managing it satisfactorily.

If there is any residue or vomiting, one or two feeds of 5% glucose solution are given, and then the strength is reduced to that which was managed, and subsequent increases in strength are made at longer intervals.

The increasing strengths given are as follows: one part of expressed breast milk and two parts of 5% glucose solution (10·5 Calories per ounce); equal parts of expressed breast milk and 5% glucose solution (13 Calories per ounce); two parts of expressed breast milk and one part of glucose solution (15 Calories per ounce); three parts of expressed breast milk and one part of 5% glucose solution (16·5 Calories per ounce); then full expressed breast milk (20 Calories per ounce).

The four pound baby starts with equal parts of expressed breast milk and 5% glucose solution on the second feeding day; this is increased a strength per day. If he is a sick baby, it is wise to start him with the weaker feedings of one part of expressed breast milk and three parts of glucose solution. If the baby is very jaundiced, it is advised to keep him on the weaker strengths till the jaundice lessens, so that he will be hungrier and will take larger feedings than if given stronger mixtures.

The five pound babies are often very hungry, and they are also given equal parts of expressed breast milk and 5% glucose solution on the second feeding day. They often take quite large quantities.

As stated before, it is usually safe to "appetite feed" the premature babies of four or five pounds who are strong enough to take all their feeding by bottle.

The feeding scheme is set out in Table II. In the case of the small premature babies an attempt has been made to make the increases in amount of breast milk each day as even and as smooth as possible.

Feeding of Infants with Pronounced Jaundice.

If the urine is bile-stained it is particularly important that the infant's fluid intake should be adequate, and at least the size of the feeding given in the scheme. If there is a tendency to vomit, or if residue is present in the case of a baby having gavage feeding, reduce the strength of the feeding or give feedings of an equivalent amount of 5% glucose solution.

If the jaundice is severe, additional parenteral administration of fluid is advisable. This will be discussed later under the complication of kernicterus.

Addition of Protein to the Feedings.

The rate of growth of the fœtus *in utero* is relatively greater than that of the newborn full-time infant, for whom breast milk is the natural food. Breast milk is thus too low in protein content for the premature baby in his early weeks, and it is advisable to add protein. It is unwise to add protein which is not previously partly digested, and the casein hydrolysate preparations are the ones of choice—for example, "Pronutrin", "Casydrol", "Essenamine". The safest time to add these is when the infant has been having full strength expressed breast milk for a day or two. Occasionally they may cause vomiting or frequent bowel actions. A 25% solution is given, commencing with half a drachm per pound of body weight per day when "Pronutrin" is used and one drachm per pound of body weight per day with the other two preparations. This is divided evenly over the eight daily feeds. The amount is increased as tolerated till double the original amount is given. This is usually continued till the infant leaves hospital. (For recipe see appendix.) The casein hydrolysate is added also to all artificial feedings with the exception of "Lactone Syrup" feedings.

Artificial Feeding.

It is rare that the small amount of expressed breast milk necessary cannot be obtained for a premature baby in its first two weeks. In the case of an infant under four and a half pounds in weight for whom no expressed breast milk is available, it is usually advisable to start with a very easily managed feeding—for example, sweetened condensed milk 1:16 (eight Calories per ounce) in amounts according to the scheme. Gradually increase the strength as tolerated till a strength of 1:8 (16 Calories per ounce) is reached. Correspondence of caloric value of strength of expressed breast milk with glucose and sweetened condensed milk dilutions is as follows.

One part expressed breast milk, three parts glucose solution = sweetened condensed milk 1:14 (9·5 Calories per ounce).

One part expressed breast milk, two parts glucose solution = sweetened condensed milk 1:12 (10·5 Calories per ounce).

One part expressed breast milk, one part glucose solution = sweetened condensed milk 1:10 (13 Calories per ounce).

Two parts expressed breast milk, one part glucose solution = sweetened condensed milk 1:9 (15 Calories per ounce).

Three parts expressed breast milk, one part glucose solution = sweetened condensed milk 1:8 (16·5 Calories per ounce).

When the infant is well established on the sweetened condensed milk 1:8, he can be graded gradually to a permanent feed. The sweetened condensed milk is used always as a temporary expedient only. (For method of grading see appendix.)

Infants of over four and a half pounds for whom no expressed breast milk is available in the first weeks will usually manage the permanent type of artificial feeding, provided it is suitably diluted. The feeding is strengthened according to the following regime, the amounts being those given in the scheme:

First, second and third days of feeding: half-strength feeding formula (10 Calories per ounce).

If there is no gastric residue or vomiting, badly managed stool, diarrhœa or scalding of the buttocks, the strength is increased:

Fourth, fifth and sixth days of feeding: two-thirds strength feeding formula (13 Calories per ounce).

If none of the above signs are present, increase further:

Seventh day to end of fourth week: Three-quarter strength formula increasing to four-fifths strength (15 to 16 Calories per ounce).

If none of the above signs occur the feeding is then gradually increased to full strength. (For stock feeding formula see appendix.)

If any of the above signs are present, the strength of the feeding is not increased; and if dyspepsia persists with the weak strength, it is usually necessary to give sweetened condensed milk 1:12 increasing in strength as tolerated to 1:8. As the digestive powers improve, the infant is graded on to a permanent feeding.

Vitamins.

Vitamin K Analogue.—An injection of vitamin *K* analogue is given on the first, third, fifth, seventh and ninth days. If the baby is very jaundiced or if the mother has been toxæmic, it is given also on the second and fourth days.

Ascorbic Acid.—Administration of 25 milligrammes or more of ascorbic acid is begun in the latter half of the first week. Later this amount is doubled, and many writers advocate 100 milligrammes daily. Occasionally the large amounts cause urinary scalding.

Vitamin B Complex.—Administration of vitamin *B* complex is usually begun at two weeks—for example, "Vita B" half-tablet twice daily. Occasionally this causes vomiting.

Vitamins A and D.—Administration of vitamins *A* and *D* is usually begun in the third week. Preparations containing vitamin *A* may produce vomiting and loose stools in some infants. The dose should then be reduced and gradually increased. These vitamins are usually given in the form of the multivitamin concentrates, and the additional vitamins *C* and *B* contained therein are advantageous. There are numerous satisfactory preparations—for example, "Infant Pentavite", three drops six times daily, or "Abdec", two drops six times daily.

General Care.

The infants should be handled as gently and as little as possible. Oiling is not done at birth or at any routine time. It should be done only when the baby's condition permits and is done in the cot with as little exposure as possible.

The type of clothing is not of great consequence provided it keeps the infant warm and is easily put on and removed. Weighing is done once weekly if the condition permits. All nursery personnel must be free of infection, and this regulation must be rigidly enforced.

Transport of Premature Babies.

When it is necessary to transport a premature baby from the place of birth to another institution, it is advisable to move him between twelve and twenty-four hours after birth. He should not be moved at night if possible. The infant should not be fed till after he is moved.

Where premature baby ambulances are available the dangers of transport, though still present, are diminished. The ambulance temperature should be kept between 85° and 90° F. for the larger infants and between 90° and 95° F. for the smaller infants. Overheating easily occurs and should be guarded against. The ambulances are equipped with a thermometer and oxygen cylinder. The infant is transferred in his wrappings and blanket to the ambulance. A nurse should travel with the infant and should have with her a sterile mucus catheter, nikethamide, "Lobeline" and a sterile syringe.

Complications of Prematurity.

During the first four days the chief danger to life is atelectasis. From the fifth to the ninth day the danger is kernicterus, and from the tenth to the twenty-first day the danger is infection. The premature baby is therefore not securely established till he is three weeks old, and it is wise to give a guarded prognosis till then.

Atelectasis.

The great majority of premature infants who die with atelectasis will be found to have the hyaline membrane resorptive type of atelectasis. Many theories are advanced, but the Boston theory fits in best with the observed facts. Briefly, this theory is that for some reason the premature infant inhales large quantities of liquor, of which he is unable to rid himself. This liquor loses water to the circulating blood because of its hypotonicity and also loses more water in the exhaled air. The soluble protein left behind separates out in the alveolar ducts into an impermeable layer preventing the entry of air into the alveoli. The air in the alveoli is absorbed, and so the alveoli become collapsed and a progressive secondary atelectasis occurs. The clinical picture is typical. The infant at birth frequently cries and is a good colour. Any time in the first twenty-four hours, from an hour after birth, the infant will have a cyanotic attack. He may die in this first attack, or he may recover. It will then be noticed that the colour is less good than before, and that chest retraction is present. Further attacks of cyanosis occur with a worsening of the colour and an increase in chest retraction. The type of retraction is characteristic, involving the lower half of the sternum, so that it is often bowed like a pouter pigeon, and the xiphisternum appears almost to touch the spine. The respirations are exceedingly laboured and not as frequent as one sees in other types of atelectasis. If a baby gets past his first twenty-four hours without a cyanotic attack, it is generally safe to say that he will not have the hyaline membrane atelectasis. If the infant can be kept alive till the fifth day, he generally recovers from this condition.

The important thing with this type of atelectasis is to prevent it at birth, particularly in relation to Cæsarean sections of election, to birth of second twins in which the membranes are ruptured just prior to the extraction of the baby, and to hydramnios. Once the fluid is in the alveolar ducts, it cannot be sucked out.

In treatment, the first necessity is to conserve the infant's energy in every possible way. The raised respiratory rate causes an increased loss of water in the exhaled air, and a watch should be kept on the infant's hydration once any œdema present has disappeared. If the infant is unable to suck sufficient fluid, or if the use of gavage distresses him, he can be given the requisite fluid by the subcutaneous drip method.

The head of the cot should be a little raised to give the diaphragm free play. Continuous oxygen administration is necessary. Prophylactic injections of penicillin and streptomycin are given for four days. The baby should be under continuous observation for the attacks of cyanosis which occur from time to time. An injection of nikethamide, one minim per pound of body weight, is given; and if the infant does not respond, it is followed by half a millilitre of "Lobeline". The rate of oxygen flow is increased during the attack. Handling of these babies should be reduced to an absolute minimum. In some cases in which there is a persistent tachycardia with a rate of over 160 per minute, digitoxin has appeared to be of benefit.

It is probable that the attacks of cyanosis which in past years were attributed to obstruction by mucus *et cetera* were really due to this type of atelectasis.

Grunting Respiration.

Grunting respiration may be heard shortly after birth and is usually accompanied by œdema. The condition generally occurs in infants who have been anoxic at birth and have required a good deal of resuscitation. It usually clears in about twelve hours, though in very anoxic infants it may persist for as long as three days.

Physiological Jaundice.

Most premature babies show physiological jaundice, but it usually appears later than twenty-four hours after birth.

Premature Kernicterus.

In premature kernicterus the onset of jaundice usually begins before the baby is twenty-four hours old, and the urine shows mild bile-staining in the early days. The jaundice deepens, and about the fourth or fifth day becomes very marked. About the fifth day the infant begins to show signs of involvement of the central nervous system. There is some mild head retraction with wrinkling of the brows and aimless wandering of the wide-open eyes. There is often frequent wide waving of the arms. In the following days the jaundice becomes deeper and vomiting occurs. The head retraction increases, and spasticity of the limbs is present. At times instead of spasticity the infant exhibits apathy and marked hypotonia, so that he lies like a rag doll.

Death usually occurs by the ninth day. If the infant survives this period, he usually lives, but there may be evidence of central nervous system damage.

This condition is seen most often in three pound babies and is quite distinct from the kernicterus which occurs in fœtal erythroblastosis.

I think various conditions can cause intense jaundice in a premature infant and that any intense jaundice can produce this premature kernicterus. The babies who are likely to develop kernicterus are as follows: (*a*) the infant with marked anoxia at birth; (*b*) the premature infant

with marked bruising at birth; (c) the infant with a septic infection, generally acquired via the umbilical cord—this condition can cause an epidemic of kernicterus in a premature ward; (d) the dehydrated infant—dehydration causes a hæmoconcentration with a rise in bilirubin concentration, so that existing jaundice is increased, and a small urinary output, so that little bile is excreted.

The most important aspect of treatment is the preventive. Any of the infants in the four categories above or any baby who shows jaundice within the first twenty-four hours should have his urine closely watched for bile. Every endeavour should be made to get the baby to take his full amount of fluid in accordance with the scheme; and should there be vomiting or residue, an equivalent amount of 5% glucose solution is given instead. If the jaundice is marked by the third day and the urine is bile-stained, and there is no œdema, subcutaneous drip therapy is instituted.

If the absorption from the tissues is poor, it may occasionally happen that intravenous administration of fluid (5% glucose in *aqua destillata*) may be required. This should be given under close observation to prevent overhydration.

Ascorbic acid, 50 milligrammes daily, is given to prevent loss of amino acid in the urine, and vitamin *B* complex can be cautiously introduced.

Feeding of low fat content, such as sweetened condensed milk 1:16 to 1:12, is given, and casein hydrolysate solution (preferably "Pronutrin" because of its smaller bulk) is added to the feeding, starting with half a drachm per pound of body weight per day and being cautiously increased to double this amount if tolerated. If the baby can be carried to the end of the ninth day without signs of central nervous system involvement, he is usually safe.

Infection.

In the second and third week infection may occur, the commonest forms being bronchopneumonia and sepsis. These are treated on general lines. Bronchopneumonia can be treated with crystalline penicillin, 50,000 units four-hourly for two or three doses, then six-hourly and later eight-hourly. It is advised to give streptomycin also in the usual dose of 20 milligrammes per pound of body weight per day. If there is no response to this, the other antibiotics can be given instead—for example, "Aureomycin" six milligrammes, "Terramycin" 10.0 to 12·5 milligrammes, or "Chloromycetin" 20 to 25 milligrammes per pound of body weight per day. When these broad spectrum antibiotics are used, it is wise to give vitamin *B* complex orally and vitamin *K* analogue by injection, and to watch the infant closely for buccal thrush.

Infants with sepsis should be isolated and treated with the appropriate antibiotic to which the particular organism is sensitive.

Other Complications.

Other complications which may occur are intracranial trauma and anæmia. Intracranial hæmorrhage is more common in premature than in full-time infants. Because of their thinness lumbar puncture is easier in these than in full-time babies. In diagnosis we must remember that three pound premature babies have normally a large amount of cerebro-spinal fluid outside the brain, and that in premature babies the protein content is higher than in full-time babies.

Anæmia.

Most small premature infants of two or three pounds show anæmia about the fourth to the sixth week. This is of a macrocytic type and is due to the immaturity of the erythropoietic system. It is not due to iron deficiency and cannot be prevented by the giving of iron.

When the hæmoglobin level falls below nine grammes (approximately 60%), the infants show signs as a rule of lethargy and poor appetite. They are then treated by transfusion. The amount given is based on the equation:

$$\frac{100 - \text{hæmoglobin value}}{100} \times \text{blood volume}$$

The blood volume is taken at one and a half ounces per pound instead of the usual one and one-third ounces. This allows for the citrate in the transfused blood. The transfusion is given by the drip method, and the rate should not exceed as many drops per minute as the baby weighs in pounds. Besides the immediate effect of raising the hæmoglobin level, the transfused red cells provide some iron.

Transfer to Expremature Room ("De-premming").

By the time the ordinary premature infant is five and a half pounds in weight he is usually ready to be moved from the heated premature ward. The cot is unlined, and he is dressed in ordinary clothes. If he maintains his temperature satisfactorily, he is moved to the "ex-prem" ward at ordinary room temperature. Here he is bathed and is put to the breast. It is a mistake to yield too soon to the relatives' importunities and to allow the baby to go home before he is securely established either on the breast, if he is breast fed, or the bottle, if he is artificially fed. The babies are usually discharged from hospital when they weigh six pounds and are then having six feeds daily. It is generally found that this stage is reached just about the time when the baby would normally be due to be born.

If possible the infant and mother are transferred to a mothercraft home till the mother is confident of handling the baby.

Iron.

It is usual to commence the administration of iron to the infant at the age of four weeks. This is begun in small doses and continued till the infant is twelve months old. The full dose is three grains of iron and ammonium citrate, or its equivalent, three times daily.

After-Care of the Premature Infant.

In the follow-up various conditions may be encountered.

Retrolental Fibroplasia.

Retrolental fibroplasia is no longer a problem in Melbourne since the administration of oxygen has been restricted to therapeutic use only and its discontinuance has been gradual. I think that this is a preventible condition. The only fresh case I know of in the last three years is that of an infant who was born in a country hospital and later sent to Melbourne.

Umbilical Hernia.

Umbilical hernia is extremely frequent. With strapping, even herniæ of the very large intimidating variety close by about the end of five months. It is, therefore, a mistake to subject these infants to operation, at any rate in the first year.

Riboflavin Deficiency.

Riboflavin deficiency I find very common. It manifests itself by the infant's continually straining and bearing down and becoming suffused. This may continue during feeding and cause vomiting, and is often obvious at night. Straining may go on with distress for an hour before a bowel action. The stool itself is soft and could obviously cause no trouble. The condition is seen in babies having breast milk and also in those having every variety of feeding. I think that it occurs mostly in the infants who put on weight rapidly.

On examination of the infant the muco-cutaneous junction of the anus is seen to be friable, and to split like damp tissue-paper on stretching. In severe cases anal fissures may be present, usually at the 12 o'clock, 6 o'clock and 9 o'clock positions. The median raphe at the anus is often swollen and œdematous. It would appear that the splitting on attempted defæcation sets up a spasm of the sphincter, and the straining goes on till the spasm relaxes.

If the infant is not already having vitamin *B* complex, he should be given it; and if he is already having it, it should be increased in amount—for example, "Vita B" tablets, one twice daily, or "Combex", half a capsule twice daily. Riboflavin tablets (five milligrammes), one tablet five times daily for an infant under three or four months of age and double this quantity over this age, are generally effective in clearing the condition in ten days. If it has not disappeared in this time it is generally necessary to give one of the vitamin *B* complex preparations by injection, half a millilitre daily for a week and then on alternate days for another week. If the fissures have become infected, healing will, of course, take longer, and a locally applied preparation such as "Monacrin" 1:1000 in cod liver oil ointment will be necessary.

Rickets.

Rickets, which is common overseas, occurs here only in infants who have not had reasonable care and is extremely rare.

Rapid Growth of the Head.

Rapid growth of the head may occur and cause fears of hydrocephalus. It is stated (Crosse, 1952) that the circumference of the head increases one and a half inches between the thirty-second and thirty-sixth weeks of fœtal life and two and a half inches between the thirty-sixth and fortieth weeks. This normal rate of growth should be remembered. The usual increase in the circumference of the head of full-time infants is one-eighth of an inch per week from birth till the age of six months.

Iron Deficiency Anæmia.

Iron deficiency anæmia may occur any time in the latter half of the first year or in the second year, if the baby has not been given iron. It is more apt to occur also if the diet is low in protein or if the infant gains weight rapidly.

Malformations.

Malformations are much commoner in premature infants than in full-time infants, and this should be kept in mind. The heart should be frequently examined, as bruits are sometimes late in appearing.

Cerebral Damage.

Owing to the greater incidence of intracranial trauma and to the obstetric calamities (ante-partum hæmorrhage, eclampsia *et cetera*) associated with premature birth, the risk of cerebral damage should be kept in mind and the milestones of development watched. The weeks of prematurity should be added to the age to give the real developmental age, and the milestones are judged on this age and not the chronological age.

Appendix.

To Sterilize Expressed Breast Milk.

To sterilize expressed breast milk, the milk should be brought to the boil in an open saucepan and boiled for two to three minutes. When it is cool, boiled water is added to make up for the quantity of water evaporated in heating the milk.

Recipe for Casein Hydrolysate.

To prepare casein hydrolysate take one medicinal tablespoon of "Pronutrin", "Casydrol" or "Essenamine" and one ounce of water. Place in a corked bottle or lidded jar in a saucepan of water. Bring the water to the boil and simmer for twenty minutes.

Method of Grading.

To carry out grading from sweetened condensed milk feeding to permanent artificial feeding, dilute the formula so that it gives 16 Calories per ounce. Then replace one or two drachms of condensed milk feeding in each feeding daily with one or two drachms of permanent formula. Take at least seven days, preferably longer, to change over. Should any signs of intolerance to the permanent formula develop, go back to the amount the baby could manage and replace the permanent feeding more slowly.

Artificial Feeding: Stock Formulæ.

A. Cow's Milk Mixture.

To prepare cow's milk mixture (20 Calories per ounce) (known as $A1_{20}$ feeding), take milk 12 ounces, lactose four tablespoons (medicinal measure) and add water to bring up to 20 ounces. This is simmered in a double boiler for twenty minutes and then brought back, when cool, to 20 ounces with cold boiled water.

B. Equivalent Mixture of Dried Milk.

To prepare the equivalent mixture of dried milk (20 Calories per ounce) (known as $B1_{20}$ feeding), take "Lactogen" six tablespoons, one and a half teaspoons (by medicinal measure), or "Glaxo Number 2" seven tablespoons, three teaspoons (by medicinal measure), lactose three tablespoons, two teaspoons (by medicinal measure) and add boiled water to bring up to 20 ounces.

C. Lactone Syrup Feedings.

1. *Cow's Milk Mixture.*—To prepare the cow's milk mixture with lactone syrup (20 Calories per ounce), take one pint of milk on which not more than two inches of top milk are left. Bring it to the boil, and boil for three minutes. Remove the skin as the milk cools. When it is stone cold stir in two tablespoons of lactone syrup and bring it up to 24 ounces with cold boiled water.

2. *Dried Milk Mixture.*—To prepare the dried milk mixture with lactone syrup (20 Calories per ounce), take "Glaxo Number 2" 12 tablespoons, two teaspoons or "Lactogen" 10 tablespoons, and add boiled water to bring it up to 23 ounces. When it is quite cold, stir in one tablespoon, three teaspoons of lactone syrup.

Subcutaneous Drip Administration.

The tissues of the thigh are chosen; and provided asepsis is maintained and the needles are suitably protected with a sterile dressing, the needle can remain *in situ* up to three or four days, although they are usually changed after forty-eight hours. A single needle can be used or a needle inserted into either thigh, a glass "Y"-piece being used. The tubing is strapped to the baby's leg, the piece nearest the needle being left uncovered for the injection of the hyaluronidase.

A small cardboard splint behind the knee is sometimes helpful. The fluid is dripped in at the rate of two or three drops per minute—that is, six to nine ounces per day. (It is useful to remember that a rate of eight minim drops per minute is equivalent to one ounce per hour.)

Hyaluronidase is injected into the tubing as near as possible to the needle at the beginning of the infusion. It is generally necessary to inject it at twelve to twenty-four hour intervals. The contents of one ampoule of "Wydase" or "Hyalase" have been those I have used most, but there are other reputable brands.

If the tissues become tense, absorption is interfered with. The drip administration should be "clipped off" till the tension disappears. A transitory redness in the tissues can be disregarded.

The fluid most used is 5% glucose in *aqua destillata*. If the baby has been having the drip administration for over forty-eight hours and has had nothing by mouth, it is wise to change to a mixture of one part of Hartmann solution and nine parts of 5% glucose solution.

The exterior of the tubing should be swabbed with antiseptic before the hyaluronidase is injected. Great care must be taken to prevent infecting the area or abscess-formation may occur.

Addendum.

I think now it is a wise prophylactic measure to instigate subcutaneous drip therapy in all infants whose urine is uniformly bile-stained, provided the infant is not œdematous. Hartmann's solution diluted in strength 1:10 with 5% glucose solution is much better absorbed than the 5% glucose solution.

KATE CAMPBELL,
Melbourne.

References.

ÅKERREN, Y., and FURSTENBURG, N. (1950), "Gastro-intestinal Administration of Oxygen in Treatment of Asphyxia in Newborn", *J. Obst. & Gynec.*, 57: 705.

CROSSE, MARY (1952), "The Premature Baby", Third Edition, Churchill, London: 3.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

DEATH BY A NATIVE SPEAR.[1]

December, 1790.

ON the 9th of the month a serjeant of marines with three convicts amongst whom McEntire the governor's gamekeeper went out on a shooting party. At one o'clock the serjeant was awakened by a rustling noise in the bushes near him and supposing it to proceed from a Kangaroo called to his comrades who instantly jumped up. On looking about more narrowly they saw two natives with spears in their hands creeping towards them and three others a little behind. As this naturally created alarm McEntire said "Don't be afraid, I know them" and immediately laying down his gun, stepped forward and addressed them in their own language. The Indians kept slowly retreating and McEntire accompanied them about a hundred yards, talking familiarly all the while. One of them now jumped on a fallen tree and without giving the least warning of his intention launched his spear at McEntire and lodged it in his left side. The man who committed this wanton act was described as a young man with a speck or blemish on his left eye; that he had been lately among us was evident from his being newly shaved. The wounded man immediately drew back and joining his party cried "I am a dead man". While one broke off the end of the spear the other two set

[1] From "A Complete Account of the Settlement at Port Jackson", by Watkin Tench (1791). From the original in the Mitchell Library, Sydney.

out with their guns in pursuit of the natives; but their fleetness of foot soon convinced our people of the impossibility of reaching them. It was now determined to attempt to carry McEntire home as his death was apprehended to be near and he expressed a longing desire not to be left to expire in the woods. Being an uncommonly robust muscular man, notwithstanding a great effusion of blood he was able with the assistance of his comrades to creep slowly along and reached Sydney about two o'clock the next morning. On the wound being examined by the surgeons it was pronounced mortal. In the course of the day Colbee and several other natives came in and were taken to the bed where the wounded man lay. To gain knowledge of the treatment of similar wounds one of the surgeons made signs of extracting the spear but this they violently opposed and said that if it were done death would instantly follow. On the 12th the extraction of the spear was however judged practicable and was accordingly performed. That part of it which had penetrated the body measured seven inches and a half long, having on it a wooden barb and several smaller ones of stone, fastened on with yellow gum most of which owing to the force necessary in extraction were torn off and lodged in the patient. The spear had passed between two ribs and had wounded the left lobe of the lungs. He lingered until the 20th of January and then expired. On opening the corpse it was found that the left lung had perished from suppuration, its remains adhering to the ribs. Some pieces of stone which had dropped from the spear were seen but no bark of wood.

Correspondence.

THE "BRITISH MEDICAL JOURNAL" AND THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

SIR: I think that the automatic receipt of the *British Medical Journal* every week by every member of the Australian Branches of the British Medical Association is now an arrangement which should be reviewed. My feelings are, and always have been, in favour of maintaining every possible link with the Old Country. But nowadays the pressure of time, cost of living and multiplicity of journals make it (at least in my opinion) desirable that subscription to the *British Medical Journal* should be optional in Australia. Many would no doubt continue to subscribe to the journal. But two good effects of this suggested adjustment would be that a good deal of economy would be effected, and the annual subscription to the British Medical Association in Australia could be reduced by a proportionate amount.

Yours, etc.,

5 Warwick Street, Killara, New South Wales. July 9, 1954. C. R. R. HEXTABLE.

THE ENIGMA OF THE MONA LISA SMILE.

SIR: Frank Trinca, of Melbourne, in his fascinatingly interesting essay (M. J. AUSTRALIA, August 14, 1954), has given a penetrating analysis and synthesis of the dual and antagonistic elements in the features of the Florentine beauty, Mona Lisa del Gioconda, as portrayed by that versatile genius, Leonardo Da Vinci. Dr. Trinca has utilized, to illustrate and emphasize the existence of opposite factors in all nature and particularly in human personality, an ingenious vertical bisection of Da Vinci's portrait of Mona Lisa.

It occurs to me that, in addition to the general principles so ably and attractively set out by Dr. Trinca, there may be a more specific and personal reason for biphasic emotional reaction in the case of Leonardo Da Vinci. Leonardo had two "mothers". The first, his real mother, a humble peasant girl, Caterina, unwed to Leonardo's father, was forced to give up the little son she loved when his father married the aristocratic Donna Albiera. At that time Leonardo was about five years old.

It seems to me possible that Leonardo saw in the features of the lovely Gioconda something that stirred in him the recollection of the adoring smile of his peasant mother, Caterina. If so, that would account for the maternal and madonna-like half of Mona Lisa's face. The other half of her face, with the raised eyebrow and the cynically seductive up-curl of the lips, might represent the reaction of the mother from whom he was cruelly torn, as if she were thinking: "That's all men want—what they can get."

Yours, etc.,

135 Macquarie Street, Sydney, August 18, 1954. ARTHUR D'OMBRAIN.

VOLVULUS OF THE CAECUM.

SIR: The article on volvulus of the caecum by John C. Fitzherbert and the letter by C. J. Windsor on appendicectomy and the McBurney incision (M. J. AUSTRALIA, August 7) are both interesting and not unrelated.

How many times has one seen a patient operated upon and an appendix not at all abnormal removed, the patient returned to the ward, and his or her subsequent history being quite unknown to the surgeon? How many of these patients still suffer from their original complaint, and seek advice from others? Has not the time come for a thorough investigation of such patients, and should we not review the work of such people as Coffey, Waugh, Carslaw, Meyers, Hemsley and others?

Dr. Windsor's remarks are also timely. No doubt the McBurney incision is a good one if the diagnosis is correct and the appendix in the usual position. Otherwise the incision recommended by Sir Hugh Devine, Professor Saint and Dr. Windsor is advisable.

Yours, etc.,

639 Sandgate Road, Clayfield, Brisbane. August 17, 1954. E. S. MEYERS.

References.

COFFEY, R. C. (1912), "The Principles Underlying the Surgical Treatment of Gastro-Intestinal Stasis due to Causes other than Structural or Ulcerative Conditions", *Surg., Gynec. & Obst.*, 15 : 365.

WAUGH, G. E. (1920), "The Morbid Consequences of a Mobile Ascending Colon with a Record of 180 Operations", *Brit. J. Surg.*, 7 : 345.

CARSLAW, R. (1928), "Right-Sided Visceroptosis", *Brit. J. Surg.*, 15 : 545.

MEYERS, E. S. (1929), "Consideration of Some of the Literature and Personal Experience in the Treatment of Gastro-Enteroptosis", M. J. AUSTRALIA, 1 : 103.

HEMSLEY, J. C. (1931), "Right-Sided Visceroptosis", *J. Coll. Surg. Australasia*, 3 : 20.

THE RESULTS OF SURGICAL TREATMENT OF BRONCHIECTASIS.

SIR: May I be granted space to comment again on the question of the diagnosis of bronchiectasis by means of bronchography?

Dr. J. A. Simpson's article, "The Results of Surgical Treatment of Bronchiectasis", in THE MEDICAL JOURNAL OF AUSTRALIA of August 14, is an account of the working out of several prevalent concepts that may be stated in the following words quoted from the article: (i) "The term 'bronchiectasis' is used to describe any permanent dilatation of the bronchial tree, the condition generally being associated, of course, with infection in the walls of the dilated bronchi." (ii) " . . . before a definite diagnosis can be made, and certainly before any rational treatment can be undertaken, adequate bronchograms, outlining all segments of the bronchial tree, must be obtained. This confirms the diagnosis and reveals the extent and localization of the disease."

If bronchograms do reveal the extent and localization of the disease, one is at a loss to understand why "good results" were obtained in only 52 of the 94 patients qualifying for review. In 13 cases readers are left in no doubt that the symptoms were not ameliorated, so that much diseased lung tissue must have been left *in situ* at the time of operation. In the remaining 29 cases, in which the results were described as "satisfactory", it is indicated that the patients "still have some symptoms". Is it too much to presume that in many of these all the diseased tissue has not been removed?

These surgical results are incongruous with the belief that bronchograms reveal the extent and localization of the diseased tissue which it is intended to remove. The observed results are easily explained by means of previously described findings (M. J. AUSTRALIA, June 12, 1954) in which chronic inflammatory damage was noted in lung fields not containing dilated bronchi as well as in lung fields containing dilated bronchi. As would be expected, bronchography has shown itself incapable of defining the extent and localization of diseased lung.

Yours, etc.,

The Royal Newcastle Hospital, Newcastle, New South Wales. J. R. S. DOUGLAS.

August 19, 1954.

THE COLLEGE OF GENERAL PRACTITIONERS POST-GRADUATE WEEK-END.

SIR: The New South Wales Regional Faculty of the College of General Practitioners is to hold its first post-graduate week-end at Dormie House, Moss Vale, on November 5, 6 and 7. This will be the first official gathering of the College following the first annual general meeting, which is to take place in the British Medical Association hall on September 24. Lectures will include recent advances in medicine, surgery and obstetrics. Members will be invited to take part in golf and bowls events. Also a dinner is to be held at Dormie House on Saturday evening, November 6. Members and other interested general practitioners who wish to attend the week-end are invited to bring their wives. Doctors who wish to arrange their own accommodation elsewhere may still attend the lectures at Dormie House. Members will receive full details of this course, and other medical practitioners who are interested may obtain full particulars by writing to me at 1015 Pacific Highway, Pymble.

Yours, etc.,

COLIN WARBURTON,
Acting Chairman, Post-Graduate Subcommittee.
College of General Practitioners (New South Wales Regional Faculty),
131 Macquarie Street,
Sydney.

August 12, 1954.

RUBELLA (GERMAN MEASLES, ROTHELN, RUBEOLA).

SIR: Dr. F. R. Yates (M. J. AUSTRALIA, July 24, 1954) asks whether the rubella virus of 1954 is capable of causing similar congenital lesions to that of 1938-1940. At the Victorian Eye and Ear Hospital are recorded 28 cases of congenital cataract in children born since 1941. A history of maternal rubella was not obtained from any patient born between the years 1941 and 1953 (24 cases). Of four children with congenital cataracts born since January, 1953, three had a history of maternal rubella occurring in the second or third month of pregnancy. In addition, each of the three had a congenital cardiac defect. These cases include the two previously reported by Dr. R. Lowe (M. J. AUSTRALIA, August 14, 1954).

Yours, etc.,

M. R. HARRISON,
Resident Medical Officer.
The Victorian Eye and Ear Hospital,
East Melbourne,

August 16, 1954.

Obituary.

DAVID HENRY EDWARD LINES.

WE have received from DR. W. E. L. H. CROWTHER the following appreciation of the late Dr. David Henry Edward Lines.

David Henry Edward Lines, who died at Hobart on June 12, 1954, aged eighty-seven years, had been in retirement from active practice for some twenty years. In that time a generation had grown up to whom the considerable part he had played in our profession was almost unknown. His first association with Tasmania was in 1895, when he came to Hobart as assistant to my father and lived with us in the old home, "Coreen", now the headquarters of the Returned Sailors, Soldiers and Airmen's Imperial League of Australia and about to be rebuilt. At that time the two surgeries and waiting room were on the ground floor, the bedrooms with day and night nurseries upstairs. Four of us (with Sally Haig, our nurse) slept in the latter, and facing it across the hall was the bedroom of the assistant. Very soon, at his invitation, we four each morning crossed over to his bed, to watch every detail of the daily shave (my father, like almost all his generation, was heavily bearded), and eat the fruit he had bought for us overnight. From the above the reader may form his own estimate of the character of the young, quiet, attractive person who was to remain our friend for almost sixty years. Born in 1867 at Melbourne, Lines was educated at All Saints' Grammar School, St. Kilda, and graduated M.B., B.S. in 1891. Practice at Woodend preceded his coming to Hobart, where he remained for eighteen months. Work in Melbourne followed until, a vacancy occurring at the Hobart General Hospital, he obtained the post and returned to Tasmania. A position as resident medical officer at this time was always regarded as a preliminary to private practice in the town, and in due course Lines resigned and joined Dr. E. J. Wolfhagen. His principal was then at the height of a most successful career; indeed, he was perhaps the first to employ the new Listerian methods at Hobart. Lines may be regarded as having been exceptionally fortunate in this association, and the partnership continued for several years, until, his interests having turned to more specialized work, he returned to Melbourne to study at the Eye and Ear Hospital.

Back in Hobart and with rooms of his own, he undertook general work and surgery with a strong bias to the special senses. An appointment as assistant honorary medical officer to the Hobart General Hospital followed, and when the first World War was declared he was fully established in practice.

The Principal Medical Officer, Colonel W. W. Giblin, having embarked in December, 1914, on the hospital ship *Kyarra* as commanding officer of the First Australian Casualty Clearing Station, Lines became acting Principal Medical Officer with the rank of major. Early in July, 1915, he in his turn went overseas as major on Number 2 Australian Hospital Ship, and on the termination of his service in 1917 he resumed work at 174 Macquarie Street, Hobart, which building had been built during the war.

In the immediate post-war period Lines disposed of his surgical practice and limited himself to the eye, ear, nose and throat. Ten years later (1930), on his retirement, Dr. J. B. Hamilton and Dr. B. Hiller took over these specialties.

His interest in medical affairs was maintained to the end. As a foundation fellow of the Royal Australasian College of Surgeons, he took a leading part in its activities in Tasmania and rarely missed a meeting. He was, too, a member of the Medical Council of Tasmania, being appointed to the Council in 1919 and selected as president twelve years later. In this capacity he continued until his resignation in 1937. Lines was also President of the Tasmanian Branch of the British Medical Association for a year. When in 1933 the decision was made to hold the Australasian Medical Congress (British Medical Association) of 1934 at Hobart, the question of who should be president was of first importance. The profession in Tasmania had for years been bitterly divided over the hospital dispute, and the compromise selection of Lines for the position proved to be a most happy choice. The honour gave him intense pleasure, and it was a delight to serve him and witness his great personal success.

I remember well an occasion towards the end of his active life, when Dr. Lines in the course of a speech expressed genuine surprise at the success attained in his medical career. Actually he was not academically minded and contributed little or nothing to medical literature. At the opening of the Congress in 1934 he paid a moving tribute to the late Dr. Eric Jeffrey in acknowledging the assistance received from him in the preparation of his address. What he had accomplished in life was due rather to his sterling qualities. In person he was of not more than middle height, compact in build, rugged and incisive in feature. His character matched his physique: a strong personality, simple, genial and humane; not sparing of words but never using them lightly. As a surgeon he was wise, patient and conservative, knew his own limitations and achieved excellent results. In religion sincere and constant, he had many years' service on the Diocesan Council and as synodsman and churchwarden of St. James's, New Town. Mention must be made, too, of his interest in the foundation of St. John's

(Church of England) Hospital. His influence was a very considerable factor in the decision of the Church to take over the old Homœopathic Hospital in Cascade Road and in its successful establishment. He served, too, on its committee of management for a number of years, and a ward in the hospital bears his name.

In daily life Lines had a strongly developed social sense, with a dry wit, and really enjoyed the company of others. Bowls and the garden took his afternoons, and he liked to visit his clubs, the Tasmanian and the Naval and Military, both of which he served as president.

The last years of his life had become increasingly difficult owing to the long illness of his wife and a relentless progress of his own arthritis. The end came at the age of eighty-seven years, when there passed a good companion, a wise and able surgeon and the only surviving Tasmanian member of the profession to have been in full general practice during the last decade of the Victorian age.

DOUGLAS JOHN THOMAS.

We are indebted to Dr. Keith Hallam for the following appreciation of the late Dr. Douglas John Thomas.

Douglas John Thomas, the son of the late Reverend John Thomas, B.A., was born in Bairnsdale, Gippsland, Victoria, on February 6, 1896. He was educated at Grenville College, Ballarat, Wesley College, Melbourne, and Queen's College, at the University of Melbourne. After completing two years of his medical course he enlisted in the first Australian Imperial Force, and served as a regimental sergeant major in the Sixth Field Ambulance on Gallipoli. He was recalled from the Middle East after returning from Gallipoli to complete his medical course, and served as a resident medical officer at Fairfield Infectious Diseases Hospital during his final year. Having graduated with first class honours in all subjects and the exhibition in surgery in 1918, he became a resident medical officer at the Melbourne Hospital, and then registrar, in which capacity he broadened the scope of his medical preparation in the X-ray and pathology departments. It was in the latter department that I first met Douglas Thomas, when, as a student, I went on some medical errand. The direct and appraising gaze, the affable and courteous manner instantly attracted me, and a close friendship began, which ten years later resulted in a medical association which lasted till his death on January 11, 1954.

Following his internship he gained his doctorate of medicine and became a medical clinical assistant at the Melbourne Hospital. For a short time he was a general practitioner in Canterbury with the late Dr. B. Stuart Cowan. In 1923 he began at Bethesda Hospital a practice in radiology and pathology and electrocardiology. The fundamental idea was to set up in a church hospital a composite diagnostic service for those people who did not wish to accept the charity of a public hospital, but could ill afford visits to varied investigators in Collins Street. His original equipment was in a small room. Films were hung to dry from strings across the room, and the records were kept in a chest with a lift-up lid. From these primitive beginnings the practice grew to warrant a special building provided by the Bethesda Hospital authorities in 1927. In the meantime he carried on a consulting practice as a physician and had been appointed to the staff of the Melbourne Hospital in 1924. At the time he and I became associated in practice his days and nights were very full. He started work very early—often between 6 and 7 a.m.—and his day's work went on till the late hours. During the week-ends he was travelling the country preaching in Methodist churches, and organizing young men particularly to help in founding the Methodist Babies' Home. Sparing himself not at all in the service of his church and of other people, by his late thirties he had lived a working life equal to that of most retired men.

In 1935 he went to England and attained a diploma in medical radiology. The next year he went again to England and gained his membership of the Royal College of Physicians. A Foundation Fellowship of The Royal Australasian College of Physicians followed a few years later as a matter of course.

In World War II he again enlisted and served for six years as a medical officer, his outstanding organizing ability being used to the best purpose as medical officer in charge of military hospitals.

His work for organizations was astounding. Many times in his life he was called upon to solve some knotty problem, and having cut the Gordian knot, was asked to plan so that the pattern for the future would not end in an insoluble tangle. On such occasions he would shut himself away for days at a time to concentrate on and elucidate the set problems, and finally would produce a charter imbued with good sense, sound financial principles, if these were involved, and a clarity that convinced and precluded useless antagonism.

His vision encompassed broad horizons and looked beyond them into a future with a precision that was uncanny. And yet an immediate problem that brooked no brooding could be solved by him with a bold decision that resulted in action along straight lines to the rapid completion of a task. A suggestion would be endorsed or rejected on the spot. For many in many spheres of life, the way ahead, that seemed foggy and blurred, was lighted and the goal gleamed after a consultation with Douglas Thomas. To me he never seemed happier than when given a mountainous task to conquer in the service of other people. Idleness and purposeless recreation were to him anathema because an anergic slice of life was wasteful. The energy generated by him in his comparatively short lifetime would power the lives of

three of us ordinary men who like leisure and dilatory recreation. His recreations were painting and gardening, wherein the wonders of creation were manifested, for he must create: not for him the padding of the beaten paths.

I have tried to picture this man with the dynamic personality to whom people turned when they wanted big things to go right. His full service to the Methodist Church, the British Medical Association, the Royal Melbourne Hospital, the Cancer Institute, the Red Cross and many other bodies cannot be told. He was a lay preacher in his church, the President and Treasurer of the Victorian Branch of the British Medical Association, Trustee of the Medical Society of Victoria, President of the Occupational Therapy School of Victoria, Chairman of the Cancer Institute Board, Medical Superintendent of Bethesda and Epworth Hospitals, and President of the Methodist Babies' Home.

Withal he was a most charming and pleasant man to work with, discussing at length problems that were worth discussing, and decisively dismissing without waste of time those that seemed to him to be trivial. In his later years he spoke dispassionately of the end in sight, and died as he had lived—hard at work. For the loss of this husband and father our sympathy goes out to his wife and son and daughter.

SIR VICTOR HURLEY writes: For almost the whole of his professional career Dr. Douglas Thomas was associated with the Royal Melbourne Hospital. Commencing as a resident medical officer in 1949, and later as registrar, he was elected to the medical staff as honorary physician to out-patients in 1924. He became physician to in-patients in 1939, and continued in this position until he resigned in 1953 on the grounds of ill health. During these years he was an enthusiastic and inspiring teacher who left a lasting impression on the students and residents who passed through his hands.

Few medical men have extended their activities over so wide a range, or combined to such a degree a wide professional knowledge and experience with sound business and organizing ability. He pioneered the establishment in Melbourne of up-to-date X-ray and pathology departments in association with private hospitals—a service of the greatest value to the profession. In the doing of this, problems of buildings, finance, equipment and staff were effectively solved; and in later years four such units were, with a large staff, carrying out a full range of service. He had a sound knowledge himself of the techniques in each of the various branches of this work, and could in an emergency carry on any particular aspect of it himself.

During his war service in the second World War he filled the position of commanding officer of military hospitals both in the field and the base—and was for some time the commanding officer of the military hospital at Heidelberg.

From 1945 onwards he was a member of the Committee of Management of the Royal Melbourne Hospital, where his professional knowledge and wide experience in hospital administration were most valuable. He was Chairman of the Cancer Institute Board from 1949 to 1952, and was one of the prime movers in the formation of the Peter MacCallum Clinic in Melbourne, where accommodation and modern treatment are available for patients suffering from malignant disease in its various forms.

It was known to him and to his close friends, for some time before his death, that his health was precarious, but he courageously carried on his many activities under great difficulties almost up to the time of his death. His loss will be felt widely, not only by the large numbers of the profession with whom he came in contact, but also in the many other sections of the community to which he rendered service.

DR. LEONARD BALL writes: It was my privilege to be President of the Victorian Branch of the British Medical Association during the last year in which Douglas Thomas was Treasurer and I succeeded him in that position. He was elected to the Branch Council in 1938 and was made Secretary in 1940—a position which he held for a short time until his departure overseas with the Australian Imperial Force. On his return to civil practice, he was appointed Librarian in 1947, and President of the Branch in 1949, and then Treasurer until his death in 1954. In 1949 he was appointed a Trustee of the Medical Society of Victoria. My association with Douglas Thomas, although it extended over many years, was particularly close in the post-war period, when we were both members of the executive of the Branch Council, and I had the opportunity of seeing just how great was the service he gave to our Victorian Branch and to the Association in general. He brought a keen incisive mind to our problems and was a tremendous help to us, more particularly in the reorganization of our financial affairs. The members of the Victorian Branch are grateful for the service he has given them and for the privilege of knowing him.

PROFESSOR DOUGLAS WRIGHT sends these extracts from the records of the Cancer Institute Board:

> Dr. Douglas Thomas was unanimously elected as Chairman of the Cancer Institute Board at its first inaugural meeting held on 27th April, 1949, and held this office till his retirement due to ill health in October, 1952.
>
> At a meeting on 11th November, 1952, the following resolution was passed: "The Board regrets that on medical advice Dr. Thomas has found it necessary to relinquish his membership and records that he was unanimously elected Chairman at the inaugural meeting of the Institute and retained that office until his retirement. Dr. Thomas is sincerely thanked for the manner in which he applied his untiring energy to the duties of his office. His unique experience enabled him to contribute greatly to the foundation of this Institute, and to bring to the citizens of this State a service of the highest standard and efficiency."
>
> At a meeting on 22nd February, 1954, the following resolution was passed: "It is resolved to place on record a minute expressing the appreciation of the Board for the role played by the late Dr. Thomas and the invaluable contribution made by him, which assisted greatly the substantial progress made in giving effect to the objects of the Act of Incorporation of the Cancer Institute Board."

Professor Wright said it was largely his dynamic, combined with political, ability which allowed the institution to be firmly established in times of greatest difficulty.

THE REVEREND DR. C. IRVING BENSON writes: Douglas Thomas came of parents distinguished for their piety and good works whose influence is an abiding fragrance in the house of memory. This we may say of him—that he kept the faith. He was loyal to the royal in him. He loved the Church and served her with vision and devotion. His steady, sustained maintenance of fellowship in worship of the Church was a great witness.

Take him all for all, here was a man—noble, generous, industrious, erratic, in some moods flamboyant as a Florentine, in others humble as a shriven monk—always supremely lovable.

I would not have you think, nor would you have me say, that he was light without a shadow. But as I think of him in the perspective of the years I shall always remember the antiseptic grace of penitence that was in him.

He had a supreme gift of organization, and yet beauty was essential to him. He had the vision of a true artist, and his own painting revealed remarkable talents. Sir Arthur Streeton tried to call him from his chosen career to devote his life to art.

So I think of Douglas Thomas in the realms of light, achieving what here he only attempted and painting on a nobler canvas the ideals which here he dreamed.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Annual Subscription Course, Lectures and Activities, September-October, 1954.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the following lectures and activities will be held in the annual subscription course during the months of September and October, 1954:

Mr. Rodney Maingot, surgeon at the Royal Free Hospital, London, will be visiting Sydney from September 2 to October 14 as Guest Professor to the Royal Prince Alfred Hospital. He will give the following lecture on September 16, at 8.15 p.m., in the Stawell Hall, 145 Macquarie Street, Sydney: "Abdominal Incisions and Some Complications following Abdominal Operations."

Professor Gordon King, Professor of Obstetrics and Gynæcology in the University of Hong Kong, will give the following lecture on September 22, at 8.30 p.m., in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, by courtesy of the staff of King George V Memorial Hospital in conjunction with the Section of Obstetrics and Gynæcology of the British Medical Association: "The Treatment of Eclampsia." Owing to alteration of his itinerary, Professor Gordon King will not arrive in Sydney in time to give his lecture on "Extrauterine Pregnancy" which had been arranged for September 15.

Dr. J. G. Scadding, dean and director of studies at the Institute of Diseases of the Chest, Brompton Hospital, and physician and senior lecturer in medicine at the Post-Graduate Medical School of London, will be in Sydney from September 25 to October 5. He will be conducting a course in thoracic diseases from September 27 to October 1 and giving the following two lectures at 8.15 p.m. in the Stawell Hall, 145 Macquarie Street, Sydney: September 28, "Sarcoidosis" (in conjunction with the Australian Laennec Society); October 1, "The Use and Abuse of Antibiotics in Respiratory Disease".

Special Lectures on Cancer.

Dr. George Pack, chief of the gastric and mixed tumour service of the Memorial Hospital, New York, well-known

speaker and author on cancer problems, and Dr. Brewster S. Miller, director of the professional section of the American Cancer Society, New York, will be in Sydney from September 27 to October 6.

Professor R. McWhirter, Professor of Medical Radiology in the University of Edinburgh and director of the Radiotherapy Department, Royal Infirmary, Edinburgh, will be visiting Sydney from September 27 to the end of October.

Arrangements are being made for Dr. Pack and Dr. Miller to conduct a panel discussion on cancer, together with Professor McWhirter, on September 29, at 8.15 p.m., in the Stawell Hall, 145 Macquarie Street, Sydney.

Dr. George Pack will also take part in a seminar on "Carcinoma of the Stomach" on September 30, at 4.15 p.m., in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital.

It is hoped that time will permit arrangements for talks to be given by Dr. Pack on "Problems of Pigmented Moles and Malignant Melanomas", "Sarcomas of the Somatic Tissues" and "Surgical Treatment of Tumours of the Liver", and by Dr. Brewster S. Miller on "Recent Developments of Cancer Research" and "The Early Detection of Cancer".

Public Lecture, October 5.—Arrangements have also been made for Dr. Pack and Dr. Miller to give a public lecture on "Cancer, the Incidence and Control" on October 5 at 8 p.m. in the Assembly Hall, Margaret Street, Sydney. Those wishing to have their names placed on the invitation list are asked to notify the Honorary Director of the Post-Graduate Committee.

Professor R. McWhirter's programme will include talks in the subscription course and to interested bodies on the following subjects: the importance of early diagnosis and treatment of cancer, the treatment of breast cancer, brain tumours, cancer of the thyroid, cancer of the mouth and naso-pharynx, cancer of the urinary tract, classification and treatment of bone sarcoma including tumours of lymph nodes, dysfibroplasia of bone and congenital familial dysfibroplasia of the jaws, adnexal tumours derived from skin and mucous membranes, testicular tumours, methods of presentation of the results of treatment. Fuller details will be announced shortly.

Cocktail Party.

A cocktail party will be held on September 30 from 5.30 p.m. to 7 p.m. in the Starlight Room at the Hotel Australia in honour of the visiting lecturers. The cost per ticket will be £1 1s. Those wishing to attend are requested to send their cheque to the Honorary Director, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, at an early date.

Fees.

The annual subscription course covers attendance at lectures by overseas visiting lecturers and other specially arranged activities. The annual fee is £2 2s. from July 1. The fee for resident medical officers is £1 1s.

Medical Prizes.

THE NORMAN McALISTER GREGG TRIENNIAL PRIZE IN OPHTHALMOLOGY.

THE following are the conditions applicable to the Norman McAlister Gregg Triennial Prize established by the Ophthalmological Society of New South Wales for a work on an ophthalmological subject:

1. The Ophthalmological Society of New South Wales will every three years award a prize of 100 guineas (Australian) and a bronze medallion for a work on an ophthalmological subject.

2. The prize will be awarded to the candidate whose work is deemed to be the most valuable contribution to knowledge in ophthalmology.

3. Entries for the prize competition will be received from any British subject. Entries will close for the present prize on August 31, 1955, at midday.

4. The work to be submitted by the candidate must be in the English language. The work must be original, and if such work has been submitted previously for publication, details must be supplied by the candidate.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 14, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	4(1)	2(2)	4(2)	..	3(3)	..	..	..	13
Amœbiasis	..	..	..	..	3(3)	..	..	..	3
Ancylostomiasis	..	..	..	..	..	..	1	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	1(1)	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	8(6)	2(2)	..	..	..	..	..	10
Diphtheria	3(1)	1(1)	6(5)	..	5(5)	..	..	..	15
Dysentery (Bacillary)	..	1(1)	3(3)	..	..	..	1	..	5
Encephalitis	2(1)	..	..	2	..	..	1	..	5
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	25(11)	22(8)	..	..	8(6)	..	1	..	56
Lead Poisoning	..	..	4	..	..	..	..	..	4
Leprosy	..	..	..	..	12	..	4	..	16
Leptospirosis	1	..	3(1)	..	..	..	..	..	4
Malaria	..	1	..	..	..	1	..	..	2
Meningococcal Infection	5(2)	7(4)	..	..	2(2)	..	..	..	14
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	10(5)	11(5)	..	5(2)	4(1)	..	..	..	30
Puerperal Fever	1	..	..	..	..	..	..	..	1
Rubella	..	2(2)	..	..	7(5)	..	..	..	9
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	14(8)	29(17)	10(7)	6(6)	1(1)	..	..	..	60
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	765	..	..	..	765
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	37(28)	21(18)	4(3)	4(2)	8(6)	3(2)	3	..	85
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

5. Examiners shall be appointed for the competition by the Council of the Society, and the Council's decision as to the winning entry shall be final and conclusive. In the event of the Council being of the opinion that no work is of sufficient merit it shall not award the prize.

6. Intending candidates shall notify the Secretary of the Society, Dr. E. J. Donaldson, 229 Macquarie Street, Sydney, of their intention to submit contributions at least two months before the closing date.

7. Three copies of the work are to be submitted. It is to be typewritten and double-spaced. Each work shall bear a motto and shall be accompanied by a sealed envelope containing the author's name and qualifications.

Notice.

PHYSIOTHERAPY CONGRESS.

THE Victorian Branch of the Australian Physiotherapy Association is conducting a congress in Melbourne from October 3 to 9, 1954. Physiotherapists from all State branches will be attending. Lectures and demonstrations have been arranged at the metropolitan hospitals, and the honorary medical staffs of these hospitals have been most cooperative. With their assistance combined lectures have been arranged for the evenings, as well as sectional meetings on specialized subjects during the day. Demonstrations by physiotherapists will be given in conjunction with these sessions.

BEATTIE-SMITH MEMORIAL LECTURES ON INSANITY.

THE twentieth Beattie-Smith Memorial Lectures on Insanity will be delivered by Dr. E. Cunningham Dax, Chairman of the Mental Hygiene Authority, in the Anatomy Theatre, University of Melbourne, at 8.15 p.m. on September 22 and 29, 1954. The subject will be "Social and Creative Activities in Mental Hospital Treatment". These lectures are open to all members of the medical profession, and entry will be free and without ticket.

Medical Appointments.

Dr. V. L. Hawke has been appointed honorary anæsthetist at the Queen Elizabeth Hospital, Adelaide.

Dr. W. D. Proudman and Dr. R. B. Jones have been appointed casualty registrars at the Royal Adelaide Hospital.

Dr. G. A. Lendon, Dr. F. H. Beare, Dr. E. McLaughlin, Dr. F. R. Hone, Dr. M. E. Chinner and Dr. K. S. Hetzel have been appointed to be a panel of medical officers, pursuant to Section 97A of the *Workmen's Compensation Act*, 1932-1953, of South Australia.

Dr. C. H. Hembrow has been appointed a member of the Masseurs Registration Board of Victoria.

Dr. L. T. Wedlick has been appointed a member of the Masseurs Registration Board of Victoria.

Dr. B. A. Sinclair has been appointed a public vaccinator for the Shire of Swan Hill, Victoria.

Dr. J. Davis has been appointed a member of the Board of Optometrical Registration in the Department of Public Health, Victoria.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Hoyle, Alan Ross, M.B., B.S., 1953 (Univ. Sydney), 17 Urabatta Street, Inverell, New South Wales.

Rosenzweig, Siegfried, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1953, 1 Roslyn Road, Elizabeth Bay.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Carruthers, Raymund Gerrard, M.B., B.S., 1954 (Univ. Sydney); Clark, Rodney James, M.B., B.S., 1954 (Univ. Sydney); Duncan, Howard, M.B., B.S., 1954 (Univ. Sydney); MacMahon, Robert Anthony, M.B., B.S., 1954 (Univ. Sydney); O'Halloran, John, M.B., B.S., 1954 (Univ. Sydney); Pickard, Beda, M.B., B.S., 1954 (Univ. Sydney); Rawle, John Glanville, M.B., B.S., 1954 (Univ. Sydney); Segall, Margot, M.B., B.S., 1954 (Univ. Sydney); Smith, Robert Gordon, M.B., B.S., 1954 (Univ. Sydney); Tebbutt, Robert John Limbury, M.B., B.S., 1954 (Univ. Sydney); Walker, John Edward, M.B., B.S., 1954 (Univ. Sydney); Dwyer, Brian Michael, M.B., B.S., 1953 (Univ. Sydney); Stump, Kenelm John Witherington, M.B., B.S., 1953 (Univ. Sydney); Cahill, Arthur Paul, M.B., B.S., 1941 (Univ. Sydney); Carter, Ian Dan, M.B., B.S., 1952 (Univ. Adelaide); Fuller, Kenneth Thomas, M.B., B.S., 1952 (Univ. Sydney); Glass, Kenneth David, M.B., B.S., 1952 (Univ. Sydney).

Diary for the Month.

SEPT. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 10.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 13.—Victorian Branch, B.M.A.: Finance Subcommittee.
SEPT. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 15.—Western Australian Branch, B.M.A.: General Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.